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PROGRESS IN THE TREATMENT OF SUBACUTE BACTERIAL ENDOCARDITIS*

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IT IS extremely gratifying for the medical profession to witness progress in therapy, especially when it applies to ailments that were formerly fatal. To the diseases of this kind, such as diabetes, pernicious anemia, Addison's disease and various forms of bacterial meningitis, which in the past almost always resulted in death it may soon be possible to add some types of bacterial endocarditis. Among the common diseases of the heart, none has been so resistant to treatment and so invariably fatal as subacute bacterial endocarditis. Although isolated cures occurred before the introduction of the sulfonamides, there is little evidence that any previous type of therapy materially influenced the outcome. The purpose of this paper is to summarize briefly the experience of the Peter Bent Brigham Hospital in the treatment of 347 cases of this disease during the years 1913 through 1944.

The thirty years covered by this study have been divided into three periods. During the first period, 1913 – 1937, termed "the pre-sulfonamide era," a great many therapeutic procedures were carried out. During the second period, 1937–1944, called "the sulfonamide era," various sulfonamides were employed¶; and during the last period, from January, 1944, to the present writing, termed "the penicillin era," penicillin has been used.** In the first period, 237 cases of subacute bacterial endocarditis were observed, during the second 90 cases, and during the third 20 cases.

PRE-SULFONAMIDE ERA

The extraordinary fact stands out that before the introduction of sulfonamide therapy there was not a single known recovery in this series. Of the 237 patients treated in this period, 218 died in the hospital or shortly after discharge, and the rest went home critically sick and were not heard from. Among other hospital patients a temporary cure or arrest may have occurred, since there were several cases of apparent recovery in patients with a mild course who had some of the evidences of subacute bacterial endocarditis but failed to show a positive blood culture. These cases were not included in the study.

It is interesting and somewhat humiliating to recall the variety of bizarre and fanciful methods of treatment that were tried in pre-sulfonamide days. One patient was infected with ratbite fever (*Spirillum minus*), another was given malaria, and a third was treated with typhoid vaccine shortly after fever therapy was introduced in the treatment of general pareses. A fourth patient received intramuscular turpentine to produce a sterile abscess, a fifth received intravenous bacteriophage, and a sixth had a resection of the breast with searing of the pectoral muscle, which had been advised by Bier.[1] Others were given antistreptococcus goat serum or various chemicals, such as neosalvarsan, Optochin, gentian violet and acriflavine. Finally, in 2 cases living beta-hemolytic streptococci in increasing doses were injected subcutaneously with the hope of altering the immune state of the host. In no case was the outcome altered.

SULFONAMIDE ERA

Of the 90 cases in the second period, 55 received adequate treatment with one or another of the sulfonamide drugs, and 35 either were not diagnosed soon enough or received inadequate chemotherapy. The patients who received a satisfactory course of a sulfonamide can be further subdivided into two groups. Thirty-nine received the drug alone, and 16 were given fever therapy in conjunction with it.

Among the 39 patients who received a sulfonamide without fever therapy, there were no recoveries. Courses of sulfanilamide, sulfapyridine, sulfathiazole and sulfadiazine, alone or in sequence, were

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¶In this period 5 patients with patent ductus arteriosus and bacterial endocarditis also received a sulfonamide, but these were operated on and are therefore not included in this series.

**In this period, 1 patient with patent ductus arteriosus and bacterial endocarditis received penicillin, but was operated on and is not included in this series.

used. In 2 of these cases continuous intravenous heparin was given in conjunction with oral sulfapyridine, and in 1 dicoumarin was given with sulfadiazine. Heparin therapy was discontinued because of its apparent ineffectiveness, its cost and the difficulty of administration, and finally because of a fatal cerebral hemorrhage, probably due to emboli

with the sulfonamides was their combination with fever therapy. Although great interest in the use of artificially induced fever in the therapy of bacterial disease followed the meeting of the Fever Conference in 1935,[4] the emphasis in treatment remained on the diseases in which the in vitro sensitivity of the infecting organism to elevated temperature made in vivo therapy rational.[5] The most encouraging results were obtained in gonococcal and meningococcal infections.[6] Although a few patients with bacterial endocarditis due to *Streptococcus viridans* showed temporary improvement following short courses of fever therapy,[7–9] a cure was not brought about. The additional

TABLE 1. *Summary of Data.**

Case No.	Age	Sex	Probable Duration of Infection	Dental History	Species Infecting Organism	Penicillin Sensitivity	Approximate Treatment	Result	Follow-up
	yr.					units/cc.			
1	14	F	1 mo.	Infected tooth†	*Str. viridans*	—	Sulfapyridine, 1 gm. every 4 hr. for 11 days; sulfathiazole, 1 gm. every 4 hr., with 11 infra-red treatments.	Well	4 yr.
2	25	M	19 days		*Str. viridans*	—	Sulfadiazine, 1 gm. every 4 hr., with 15 typhoid chills	Well	2 yr.
3	25	M	2 mo.	Gum infection	*Str. viridans*	—	Sulfathiazole, 1 gm. every 4 hr.; sulfadiazine, 1.5 gm. every 4 hr., with 10 typhoid chills.	Well	3 yr.
4	30	F	3 mo.		*Str. viridans*	—	Sulfadiazine, 1 gm. every 4 hr., with 9 typhoid chills	Well	2 yr.
5	20	F	1 mo.		*Staph. albus*	0.600–0.015	Penicillin, 100,000 units a day by constant intravenous drip for 10 days and 8300 units intramuscularly every 3 hr. for 9 days Sulfadiazine, 1 gm. thrice daily, maintenance dose Penicillin 300,000 units a day by constant intravenous drip for 11 days and 50,000 units every 2 hr. intramuscularly for 10 days	Relapsed in 6 wk. Relapsed in 2 mo. and died from another cause 10 mo. later	
6	52	F	3 mo. 10 mo.		*Str. viridans*	—	Sulfadiazine, 1 gm. every 4 hr., with 5 typhoid chills; sulfadiazine, 1 gm. every 4 hr. and neoarsphenamine, 400 mg., with 7 typhoid chills. Penicillin, 240,000 units a day by constant intravenous drip for 12 days	Well; subsequent heart failure.	1 yr
7	38	M	10 wk.		*Str. viridans*	—	Penicillin, 240,000 units a day intravenously for 9 days and 20,000 units intramuscularly every 2 hr. for 3 days	Died with cardiac arrhythmia 2 mo. after treatment	
8	16	F	7 mo.	Teeth cleaned and filled	*Str. viridans*	0.001	At another hospital the patient received a sulfonamide with typhoid chills and with malaria inoculata; at this hospital she received 200,000 units of penicillin a day by constant intravenous drip for 20 days.	Well	1 yr.
9	17	F	2 mo.	Tooth extractions	*Str. viridans*	—	Penicillin, 240,000 units a day by constant intravenous drip, for 16 days	Well	1 yr.
10	61	M	2 mo.		*Str. viridans*	0.080	Penicillin, 300,000 units a day by constant intravenous drip, with sulfadiazine, 1 gm. every 4 hr., for 13 days	Well; limited cardiac reserve.	16 mo.
11	56	F	3 mo.	Upper teeth pulled	*Str. viridans*	0 080	Penicillin, 300,000 units a day by constant intravenous drip, with sulfadiazine, 1 gm. every 4 hr., for 22 days	Well; further limitation in cardiac reserve.	16 mo.
12	45	M	3 mo.	Severe dental sepsis	*Str. viridans*	0.035	Penicillin, 300,000 units a day by constant intravenous drip, for 22 days	Well	1 yr.
13	27	F	2 mo.	Tooth infection†	*Str. viridans*	0.035–0.070	Penicillin, 300,000 units a day by constant intravenous drip, for 15 days Penicillin, 500,000 units a day by constant intravenous drip, for 23 days Penicillin, 20,000 units intramuscularly every 2 hr., with benzoic acid regimen, for 30 days and 20,000 units intramuscularly every 3 hr., with benzoic acid regimen, for 30 days	Relapsed in 7 days Relapsed in 2 wk. Well	9 mo.

*All patients had rheumatic heart disease except the one in Case 5, who had a postoperative infection following ligation of a patent ductus arteriosus, the one in Case 14 who had a bicuspid aortic valve and a patent ductus arteriosus and the one in Case 17, who had arteriosclerotic heart disease.

bolism, that complicated therapy in one of the above patients, as well as in another reported case.[2] The small size of this group and the rigid criteria for case selection probably account for the deviation in recovery from the usual 6 per cent rate reported elsewhere for sulfonamides alone.[3]

The most illuminating feature of our experience

finding that many strains of *Str. viridans* recovered from the blood of patients with subacute bacterial endocarditis survived and grew in vitro at temperatures above those that the patient could tolerate[7, 10] furthered the neglect of fever treatment for this disease until Bierman and Baehr[11] reported their successes in 1941. In this hospital the use of fever artificially induced fever by means of infrared radiation. It may be significant that this and other successful fever therapy cases underwent many more fever treatments than were used in the earlier failures with fever therapy alone. The first cure led to the unsuccessful treatment of 6 other patients in a similar fashion, and subsequently to the treatment

TABLE 1 (*Continued*).

Case No.	Age	Sex	Probable Duration of Infection	Dental History	Species Infecting Organism	Penicillin Sensitivity	Approximate Treatment	Result	Follow-up
	yr.					units/cc.			
14	20	M	5 mo.		*Str. viridans* *Alkaligenes faecalis*	0.001 None	Penicillin, 240,000 units a day intravenously, for 9 days and 20,000 units intramuscularly every 2 hr., for 13 days	Died	
15	32	M	4 mo.	Infected tooth filled	*Str. viridans*	0.035	Penicillin, 300,000 units a day by constant intravenous drip, for 14 days	Relapsed 4 mo. later; died of ruptured spleen.	
16	41	F	1 mo.	Infected tooth†	*Str. viridans*	0.035	Penicillin, 300,000 units a day by constant intravenous drip, for 20 days	Died 3 wk. after treatment	
17	73	M	1 mo.	No teeth	*Str. faecalis*	0.035	Penicillin, 20,000 units every 2 hr. for 15 days, and every 3 hr. for 15 days, with benzoic acid regimen	Died 3 wk. after treatment of coronary occlusion	
18	62	M	10 days		Type 1 pneumococcus	—	Penicillin, 300,000 units a day intravenously, for 12 days, 20,000 units intramuscularly every 2 hr., with 7.5 gm. of benzoic acid thrice daily, for 7 days and 20,000 units intramuscularly every 2 hr., with 7.5 gm. of benzoic acid thrice daily and low-salt and low-fluid intake, for 30 days Rest period for 15 days	Relapsed; rise in temperature, hematuria, anemia, rising sedimentation rate and negative blood culture.	
							Penicillin, 20,000 units intramuscularly every 2 hr. except 4.00 a.m., with benzoic acid and low-salt and low-fluid intake, for 26 days	Well; acute congestive failure 10 wk. later; good response to digitalis.	7 mo.
19	27	M	2 wk.		*Str. viridans*	0.007	Penicillin, 300,000 units a day intravenously for 6 days (blood level 0.07 units per cc.), 500,000 units a day intravenously for 7 days (blood level 0.14 units per cc.) and 900,000 units a day intravenously for 9 days (blood level 0.20 units per cc.)	Well	7 mo.
20	19	F	4 wk.		*Str. viridans*	0.060	Penicillin, 20,000 units intramuscularly every 2 hr. for 9 days (blood levels 0.14, 0.14 and 0.07 units per cc.), 40,000 units intramuscularly every 2 hr. for 7 days (blood levels 0.14, 0.14 and 0.07 units per cc.) and 80,000 units intramuscularly every 2 hr. for 6 days (blood levels 0.28, 0.28 and 0.14 units per cc.), all with benzoic acid, 4 gm. thrice daily, and water limited to 1500 cc. and salt to less than 2 gm. daily	Well	8 mo.
21	47	M	3 wk.	Abscessed tooth on physical examination	Alpha-hemolytic streptococcus	0.035	Penicillin, 25,000 units every 2 hr. intramuscularly for 28 days, with benzoic acid, 9.0 to 12.5 gm. daily and low-fluid and low-salt intake	Well; abscessed tooth removed during therapy.	6 mo.

†Painful obvious dental infection within 2 to 6 weeks before onset of present illness.

therapy was begun because of the obvious improvement in one patient (Case 1) after drug fever of three days' duration had occurred during a course of sulfapyridine therapy (Table 1). Despite the fact that the responsible organism grew in the presence of 5 mg. per cent sulfathiazole for twenty-four hours at 40° C., a cure followed a course of of 9 more patients with typhoid vaccine as outlined by Solomon,[12] 3 of whom (Cases 2, 3 and 4) recovered. Through the stimulation of Osgood's[13] report, 2 of the patients listed above who received a sulfonamide without fever therapy were also given neoarsphenamine, and 1 patient (Case 6), treated with typhoid-vaccine fever and a sulfonamide, was later

given typhoid vaccine, sulfadiazine and neoarsphenamine simultaneously. No one of these last 3 patients was cured by such desperate measures, whereas 1 (Case 6) was subsequently readily cured with a short course of penicillin.

When fever was induced by infra-red radiation, the rectal temperature was kept at 105 to 106°F. (40.6 to 41.1° C.) for three to five hours for eight or more treatments on alternate days. For typhoid-vaccine fever, 25,000,000 typhoid-paratyphoid organisms were injected intravenously for the first dose, and the amount was doubled at intervals of two or three days. The purpose was to obtain a temperature of 104°F. (40.0° C.) for several hours. Ten or twelve such treatments were usually given. In some cases the amount of vaccine was increased or decreased because the response was insufficient or excessive. Of the 16 patients who had this combination of fever and sulfonamide therapy, there were cures in 4 (Cases 1, 2, 3 and 4)* — the first such cures ever obtained in this hospital. These recoveries with combined fever and sulfonamide therapy are in accord with the ratio 4:25 reported elsewhere for hyperthermia[3] and that of 5:17 for typhoid-vaccine fever and sulfonamides.[13]

Penicillin Era

Of the 20 cases treated in the third period, 17 (Table 1) received adequate courses of penicillin, 2 were diagnosed post mortem, and in 1 no treatment was given because the patient was in terminal uremia on admission and the limited drug supply at the time did not justify treatment of an apparently hopeless case. Penicillin was given for two to four weeks in a constant intravenous drip of 5 per cent dextrose at the rate of 240,000 to 500,000 units a day. At the time of this writing, six to eighteen months following treatment, 11 of the 17 adequately treated cases are cured or arrested and 1 patient (Case 5) has been cured but has died of another cause. Three of these 11 patients (Cases 5, 13 and 18) required more than one course of penicillin. In 2 of the 17 adequately treated cases (Cases 10 and 11), sulfadiazine was given with penicillin. In a word, in 11 of the 17 well authenticated cases of subacute bacterial endocarditis the patients have responded favorably to penicillin therapy and have remained well for six to eighteen months.

Each of the 6 patients who died after adequate penicillin therapy presented a complicated problem. One (Case 7) died of congestive heart failure two months after penicillin was stopped. In the interval between treatment and death, auricular fibrillation, sweating and a rapid heart rate were present. During this time the patient remained afebrile and the blood cultures were sterile. Unfortunately, the cause of death is not known, for the patient died at home and no post-mortem examination was made.

The second patient (Case 14) died of general septicemia three weeks after penicillin treatment was stopped. Repeated blood cultures before treatment contained *Str. viridans*. When penicillin was begun this organism disappeared from the blood, and it was not recovered again during life or from the post-mortem heart's blood. After a week of penicillin, a gram-negative bacillus, *Alkaligenes faecalis*, appeared in the blood cultures and persisted until death, despite further penicillin and later sulfadiazine therapy. At autopsy, both *Str. viridans* and *Al. faecalis* were cultured from vegetations found on a bicuspid aortic valve and a patent ductus arteriosus.

The third patient (Case 16), with a short illness had a positive streptococcus skin test on admission that became negative before treatment and during active bacteremia. The only clinical response to penicillin given by constant intravenous drip was control of embolic phenomena and bacteremia. High fever and debility persisted. After penicillin was discontinued, the blood cultures remained negative until death three weeks later. In this interval dermatitis exfoliativa and acute yellow atrophy appeared. At autopsy healing vegetations were found that when crushed and cultured yielded the same type of streptococcus as that found in life. In retrospect this patient was probably inadequately treated. We have since found it necessary to raise the penicillin dose by steps to well over 1,000,000 units a day to control systemic symptoms of the septicemia. When even this dose is inadequate, the addition of the benzoic acid regimen gives blood levels equivalent to a daily dose of 6,000,000 to 10,000,000 units a day.

The fourth patient (Case 15) responded favorably to a two-week course of 300,000 units of penicillin a day by constant intravenous drip. He remained well until four months later, when a splenic infarct took place and a low-grade fever appeared. Death from a ruptured spleen occurred two weeks later during an interval in which repeated negative blood cultures delayed the reinstitution of penicillin therapy. At autopsy, healed and healing vegetations were found that contained *Str. faecalis* by smear and culture. The splenic infarct was sterile. The infecting organism was different from the one recovered during the first illness, and this fact invites speculation whether the patient died of a reinfection rather than of a recurrence. A ruptured spleen in subacute bacterial endocarditis is infrequent.[14–16] This case will be reported in detail elsewhere.[17]

In the fifth case (Case 17), death occurred suddenly three weeks following thirty days of penicillin therapy — 20,000 units every two hours day and night intramuscularly for the first fifteen days and

*Another patient recovered with sulfadiazine and typhoid-vaccine therapy but was not included because blood cultures were always negative, although he showed many of the other clinical features of bacterial endocarditis.

20,000 units every three hours for fifteen days more. With this schedule the salt intake was restricted to less than 3 gm. a day, fluids were restricted to less than 1500 cc. a day, and 2.5 gm. of benzoic acid in capsules was given three times a day with meals. During therapy the patient remained on the benzoic acid regimen.[18] At autopsy a healing vegetation measuring 1 cm. by 3 cm. by 0.1 cm. was found firmly attached to the closure line of an aortic cusp. It contained no bacteria on smear, culture or microscopic examination of sections. The cause of death was acute coronary occlusion, possibly due to temporary obstruction of the left coronary orifice by the curtainlike vegetation.

The sixth death (Case 5) occurred in May, 1945, following a massive hemorrhage from a bronchus. The endocarditis had developed as a postoperative complication of an unsuccessful ligation of a patent ductus arteriosus. This hemorrhage occurred seventeen months after the first course of penicillin and nine months after all evidence of infection had disappeared. Autopsy revealed the original patent ductus arteriosus opening into a large aneurysmal sac in the pulmonary artery and a silver clip eroding a small branch of the pulmonary artery and the adjacent bronchus. Apparently one of the clips used in the ligation had been engulfed by the subsequent aneurysm and had been carried by the blood stream to the lung. There was complete healing of the mediastinum and vascular walls.

Two of these deaths (Cases 5 and 17), were in cases of proved bacteriologic cure, and in 1 (Case 7) the patient may have been free of vegetations.

Dental Aspects

Although isolated cases of subacute bacterial endocarditis following tooth extractions have been known for some years, it is only recently that the great significance of this factor has been appreciated. That bacteremia follows tooth extractions in 70 per cent of cases,[19] frequently follows minor dental manipulations and may occur after the simple act of chewing has been established.[20] In this series of 347 cases the perfunctory dental histories of the routine records have revealed an incidence of tooth extractions in 10 per cent of the cases and other dental manipulations in an additional 10 per cent within several weeks before the onset of bacterial endocarditis. During the past two years, in which there has been a deliberate inquiry about dental sepsis as a part of the patient's present illness, its real importance has been demonstrated (Table 1). Among earlier patients numerous other foci of infection and a variety of surgical procedures have similarly acted as the origin of this infection — namely, tonsillectomy, sequestrectomy, abdominal operations (appendectomy, colostomy, tubo-oophorectomy and so forth), prostatic massage and others. Nevertheless, dental sepsis is the most prominent offender. The significance of this factor is emphasized by bacteriologic differentiation of the causative organisms. Most edentulous patients who develop bacterial endocarditis are infected with the enterococcus, the staphylococcus or even the gonococcus, whereas the overwhelming majority of patients who still have their teeth are infected with *Str. viridans*, which is so generally associated with dental sepsis and the bacteremia attending dental procedures. It is because of these findings in patients with subacute bacterial endocarditis who need dentistry that we have completed necessary dental extractions and manipulations while the patients have been receiving penicillin, and have continued therapy until gum healing is complete. When the teeth are so poor as to constitute a subsequent threat of reinfection of the heart valves, we have removed all of them before completing treatment.

Streptococcus Skin Tests

A final point of interest is the skin reaction to intradermal injections of streptococcus nucleoprotein. It is known that 85 per cent of cases of rheumatic fever show a positive reaction to beta-hemolytic streptococcus skin tests.[21] It is the finding of others[22, 23] and it has been our experience[24] that cases of subacute bacterial endocarditis rarely show such positive skin reactions. Post-mortem evidence of concurrent active rheumatic carditis and subacute bacterial endocarditis is not necessarily a contradictory finding, for there is reason to believe that the rheumatic state can be activated by or follow and need not precede the bacterial endocarditis.[25]

At present the streptococcus skin test is a useful empirical diagnostic tool. Seventy-four of 77 patients with undoubted bacterial endocarditis have had negative beta-hemolytic streptococcus skin tests, and all 77 have had negative alpha-hemolytic streptococcus skin tests.* Eleven patients who had negative skin tests during active bacteremia developed positive alpha-hemolytic and beta-hemolytic streptococcus skin tests, either during or shortly after penicillin therapy, and have so far maintained their positive reactions.

Discussion

It is fair to point out that all but 1 of the deaths following penicillin therapy were due to causes beyond the control of this drug. The successes, although not so fraught with bizarre complications as were the fatal cases, nevertheless were gained despite chronicity of the disease and advanced age, great debility and poor cardiac reserve of the patients. One patient (Case 6) developed congestive heart failure and had a cerebral hemorrhage or embolus during an unsuccessful course of fever and sulfonamide therapy several months before penicillin

*Details of these data will be presented elsewhere in a subsequent publication.

was administered. Although now ambulatory, she is on constant digitalis therapy and suffers primarily from moderate congestive failure. Another patient (Case 10) was in congestive failure when the diagnosis of bacterial endocarditis was first made. Since recovery he has had two bouts of mild congestive failure without evidence of infection. A third patient (Case 8) was of interest because she showed evidence of acute nephritis before treatment. The urine contained considerable albumin and many red cells, white cells and casts. There was marked anemia, with a red-cell count of 2,600,000. Despite the severity of the illness, the response to treatment was most satisfactory. A fourth patient (Case 15) did well up to his recurrence or reinfection four months later, although before treatment he had had a cerebral embolism with hemiplegia. Functional recovery of the affected side of the body was almost complete. One patient (Case 2) treated with sulfadiazine and fever and 6 patients (Cases 5, 9, 10, 14, 18 and 20) treated with penicillin had arterial emboli after therapy had been stopped, but despite these did well. The inference to be drawn is that sterile emboli may occur without preventing a favorable outcome. It is also important that the physician be not unduly disturbed at unexpected rises in temperature during the course of treatment, because these are often due to local phlebitis at the site of the constant intravenous injection. He should, however, be concerned by a continuous fever during therapy, because this usually means inadequate amounts or methods of giving penicillin — as in Case 16. Another point of considerable significance is the fact of recovery in several cases even though treatment was begun many months after the onset of the disease and in spite of the patients' advanced age. It is obvious that the outlook is better when the diagnosis is made promptly, but it is comforting to know that favorable results can be obtained in patients who have been neglected for six months or more, as in Cases 6 and 8.

Studies with the streptococcus skin tests were of special interest in the penicillin-treated patients. At the present time one may speculate that whatever the state of immunity or sensitivity of the host to streptococcal infections before the development of bacterial endocarditis, the bacteremia effectively desensitizes as well as immunizes the host during infection. The prompt control of septicemia and splenomegaly and the early appearance of a positive streptococcus skin reaction during or following penicillin therapy suggest that the principal effect of this drug is to localize the valvular infection. The finding of viable streptococci in the valves in fatal cases that had received relatively short courses of penicillin (Cases 14, 15 and 16) and not in a patient treated for one month (Case 17) suggests that at least two, often three or four and sometimes even eight weeks of penicillin — as in Case 13 — are necessary before the vegetations heal completely. Recurrent sterile emboli appearing during and shortly after treatment give further support to this theory. In the fatal case with a curtainlike vegetation (Case 17) the patient might well have had a sterile embolus during convalescence had he not died of another condition.

The use of large doses of penicillin in the treatment of subacute bacterial endocarditis was introduced by Loewe.[26] The aim of therapy is the maintenance of a penicillin blood level considerably in excess of the minimal inhibiting concentration for the patient's organism for an extended period of time. A higher level maintained for a short time does not seem to be an effective alternative mode of treatment; Case 13 is an example. Two hundred thousand or 300,000 units a day given either by constant intravenous drip or by two-hourly intramuscular injections day and night may serve to maintain such a level in the blood stream most of the time,[27] and do so continuously when combined with the benzoic acid regimen.[18] Patients with a free aortic regurgitation may require as much as 1,000,000 units a day to maintain equivalent blood levels. The patient in Case 19 was treated by the constant intravenous drip method and the one in Case 20 with the benzoic acid regimen and intramuscular injections every two hours. In the latter patient it was found to be practical to give up to 80,000 units in 2 cc. of saline solution, and this was done without any more local discomfort than is experienced with the usual dose of 20,000 units in 4 cc. of saline solution. Despite these doses, the blood levels did not exceed fivefold the minimal penicillin concentration that inhibited their respective organisms.

Because of the importance of individualizing the treatment of subacute bacterial endocarditis, whenever possible we have determined the penicillin sensitivity of the patient's organisms and gauged the necessary amount of therapy by the lowest penicillin blood levels attained in treatment. From the cases covered in this report it appears that a blood level that remains just above or often falls below the minimal inhibiting concentration for the bacteria does not control symptoms or temperature. A good working rule is to maintain a blood level five or more times the minimal effective level. Just how this is to be done varies with the patient. By the same token, no set rules for the duration of therapy can be stated. In Case 13 the patient relapsed twice, both times after apparently adequate therapy. When therapy was continued the third time until the sedimentation rate had fallen close to normal, she recovered. The patient in Case 18 relapsed but responded to retreatment which was continued until after the microscopic hematuria was controlled. The sedimentation rate was not normal but became stabilized at a somewhat elevated level. Penicillin was discontinued

in other patients when the pattern of their illness was favorable as judged by symptoms, temperature, a rise in hemoglobin, a fall in the sedimentation rate and clearing of the urine. The change of the streptococcus skin reaction to positive usually occurred before treatment was discontinued and was interpreted as a favorable sign. One patient (Case 13), however, relapsed after such a change, and 1 (Case 19) did not have a positive reaction until some weeks after treatment but nevertheless recovered. In this series recovery has followed two weeks of treatment in some cases, as in Case 6, and as long as two months of treatment in others, as in Case 13.

With special attention to the penicillin dosage in subacute bacterial endocarditis, it should be possible to raise the recovery ratio to an even higher figure than that of 11:17 in this study, especially when the disease is recognized early, the organism is found to be sensitive to a certain level of penicillin and the dosage is planned accordingly.

Summary

Three hundred and forty-seven proved cases of subacute bacterial endocarditis treated at the Peter Bent Brigham Hospital from 1913 to 1945 have been studied. There were no known recoveries among 237 cases seen between 1913 and 1937, during which time a great variety of therapeutic procedures were employed.

Of the 90 cases observed from 1937 to 1943, 55 received adequate treatment with a sulfonamide. Sixteen of the latter patients were given simultaneous fever therapy. Of these, 1 treated by infrared radiation and 3 treated by typhoid vaccine recovered and have remained well for two to four years. None of the other sulfonamide-treated patients recovered.

Of 20 patients studied since January, 1944, 17 were given large doses of penicillin. In 11 cases the infection has been cured or arrested, and the patients are now alive. The follow-up periods vary between six and eighteen months. In two others the infection was bacteriologically arrested but death occurred some weeks later because of complications.

Congestive heart failure may follow recovery from the infection.

Dental sepsis, extraction of teeth and dental manipulations were found to be related to the onset of this disease in a surprisingly large number of cases. Infected teeth should be removed before treatment is completed, as a prophylaxis against subsequent reinfection of the heart valves.

A change in the sensitivity of the skin to streptococci was found to vary with the stage of illness. The reaction was almost invariably negative during the active stage of infection (74 of 77 cases) and became positive in the patients who recovered (11 cases).

Although penicillin appears to be a promising cure for subacute bacterial endocarditis, patients who recover from this disease are left with the same or further valvular deformities, which continue to predispose them to reinfection or to subsequent congestive heart failure.

The successful treatment of a patient with subacute bacterial endocarditis is an individual problem that requires careful attention to the details of therapy. Successful treatment depends on maintaining penicillin blood levels considerably in excess of the minimal inhibiting concentration of penicillin for the patient's organism.

Abstracts of the 4 cases successfully treated with sulfonamides and of 14 of the 20 cases treated with penicillin will appear in the reprints of this paper.

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ACUTE OBSTRUCTION OF THE SMALL INTESTINE*

A Report of One Hundred and Eighteen Cases

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HOSPITALS devoted to the care of charity patients are not usually expected to be sources of low mortality rates. The reasons for this, which are many and varied, include the generally poor state of nutrition among these patients, their advanced physiological age as compared with the chronological age and, possibly most important of all, the educational deficiencies that bring them to medical care late in the course of their diseases. When these handicaps are compounded with an illness that in the best of hands carries a formidable mortality, depressing mortality figures are to be expected.

TABLE 1. *Mortality Rates in Reported Cases of Acute Obstruction of the Small Intestine.*

YEAR REPORTED	AUTHOR	NO. OF CASES	MORTALITY
			%
1930	Holden[4]	—	19.0
1938	Scudder[5]	2150	24.0*
1938	Wangensteen[6]	157	17.9
1940	Schlicke, Bargen and Dixon[7]	133	21.8
1940	McKittrick and Sarris[8]	136	20.0†
1942	Dennis and Brown[9]	48	13.3
1943	Dennis and Brown[10]	110	15.5
1944	Dennis[11]	53	15.1

*Viable bowel only.

†Hospital mortality; operative mortality, 18 per cent.

Before what might be called the "Wangensteen era," the last decade or so—the prognosis of acute intestinal obstruction was truly grave. Miller[1] reported a mortality of 61 per cent in 1929. Cole[2] states that the mortality of two decades ago was 30 to 40 per cent. Bennett[3] states that a 70 per cent mortality prevailed in earlier days. Moreover, a survey of the more recent literature reveals that this condition is still one of the most serious seen by the surgeon.

It appears from an analysis of such results as are reported that a rate of about 20 per cent is a reasonable goal at which to aim. To illustrate this consensus, the mortality rates from some leading clinics are presented in Table 1.

Richardson[12] indicts acute obstruction for causing half as many deaths as its far more frequent competitor, acute appendicitis with sequelae, and assigns to it a mortality of 40 to 60 per cent in the United States in 1944. Colcock[13] terms it "the most serious lesion affecting the small bowel." Stone and Owings[14] write that the results of its treatment are unsatisfactory and that the mortality is unnecessarily high. Dennis and Brown[10] in 1943 summarized the general mortality up to the time of their report as 18 to 20 per cent in skilled hands. Thus, the gravity of this problem is emphasized directly or indirectly by all authors, and it is undeniable that the problem of management of these patients is far from solved. The tragedy of such results must be even more apparent when it is realized that many patients represent the final harvest of trivial, even at times unnecessary, previous exploratory operations.

During the last fifteen years great forward strides have been made in the management of these problems. These have been accomplished through such procedures as the Wangensteen[6, 15] suction method, the Miller–Abbott[16] intubation method, Fine's[17] oxygen decompression method and the liberal use of parenteral fluids, blood and plasma,[2, 6, 8, 13, 18–20] to mention the more dramatic advances.

TABLE 2. *Summary of Data.*

TYPE OF LESION	NO. OF CASES TREATED	DURATION OF SYMPTOMS RANGE	DURATION OF SYMPTOMS AVERAGE	AVERAGE AGE	AVERAGE WHITE-CELL COUNT	AVERAGE PREOPERATIVE PULSE RATE	AVERAGE TEMPERATURE	CASES WITH FLUID LEVELS
				yr.	$\times 10^3$		°F.	%
Hernia:								
Umbilical	8	4 hr.–6 days	50 hr.	48	17.0	96	100.2	50
Inguinal	24	3 hr.–9 days	14 hr.	44	16.0	98	99.8	67
Femoral	5	14 hr.–6 days	28 hr.	69	11.0	70	98.0	—
Postoperative ventral	3	6 hr.–4 days	22 hr.	41	9.0	116	99.4	67
Internal	5	7 hr.–4 days	40 hr.	39	13.0	100	100.8	100
Postoperative adhesions	44	3 hr.–9 days	38 hr.	37	12.0	106	101.4	93
Inflammatory adhesions	9	10 hr.–14 days	32 hr.	32	18.0	110	101.4	89
Intussusception	5	3 hr.–38 hr.	16 hr.	1	9.0	?	100.8	—
Volvulus	5	1½ hr.–30 hr.	18 hr.	49	21.0	118	101.2	50
Mesenteric vascular occlusion.	3	2½ hr.–5 days	58 hr.	67	23.0	104	97.8	100
Carcinoma:								
Metastasis	2	15–24 days	8 days	53	7.0	76	98.6	100
Primary malignancy	1	34 hr.	34 hr.	50	6.0	82	98.0	100
Meckel's diverticulum	1	16 days	16 days	53	13.0	102	99.6	100
Regional ileitis	2	18 hr.–36 hr.	27 hr.	36	8.0	90	98.2	100
Tuberculosis of ileum	1	3 days	3 days	41	7.0	88	98.0	100
Total	118							
Averages			64 hr.	46	15.6	102	100.3	85

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Each of these valuable adjuncts, however, has been enthusiastically ushered in, only to be later ranked as falling far short of being the unmixed blessing it seemed at first. Certainly, few can deny that the efficient care of obstructed patients calls for nicety of judgment, assiduous and diligent attention and individualization hardly demanded by any other group. Such cases do not lend themselves to standardization by any known therapeutic scale. It is with this precept in mind, rather than varying formulas of duration of obstruction, objective findings of abdominal tenderness, rigidity and the like or white-cell counts, that one should attempt to treat these patients.

Any series of acute intestinal cases with an unusually low mortality of necessity demands an accounting of the methods of diagnosis employed. Little question of diagnostic accuracy is encountered in the patients receiving operations, the finding in whom of obliteration of a section of bowel lumen, with dilatation of the bowel above and its collapse below the point, eliminates argument concerning classification. Justifiable critical scrutiny, however, may be aimed at the cases not operated on. Only one of our patients with hernial obstructions was not operated on, the ultimate diagnosis being confirmed at autopsy. This method of confirmation was applied to a case of obstruction due to a metastatic malignancy and to another due to mesenteric vascular occlusion. With the exception of these cases and of those due to adhesions, all our diagnoses were confirmed at operation. Concerning the cases due to adhesions, the presence of complete obstruction may be difficult to decide with certainty without operation, and many patients who were treated conservatively have been omitted from this survey because there was a lack of one or more absolute criteria for the conclusion that obstruction was complete. Of this group of patients, 22 satisfied our criteria by the demonstration of intermittent, cramping abdominal pain, repeated vomiting, failure to pass gas or feces by rectum prior to admission and after two or more irritating enemas, typical peristalsis and roentgenologic demonstration of distended bowel, with fluid levels and absence of colonic gas. Failure to demonstrate any one or more of these diagnostic points has excluded many cases from the series.

Paramount in the criteria for judging the method of handling these problems is an assessment of the general condition rather than the local disease. We fear, as do all surgeons, the progression of bowel changes from obstruction to strangulation and finally to loss of viability, but we also believe these to be relatively overemphasized. We are convinced that it is better to operate on many of these patients six, eight or ten hours later, when they can tolerate the procedure, than to perform a possibly simpler and easier operation at once when they are too ill to stand it. We believe that it is safer to consider every patient with intestinal obstruction as critically ill and as a truly poor-risk patient, one who is actually too ill to withstand the operation that he nonetheless may require. Therefore, we do not allow our natural fear of local bowel change to carry the same weight as concern for the patient's general condition. The former is more obvious, more easily demonstrated and more readily evaluated, but the latter constitutes the greater threat to life.

Our method of treating acute obstruction of the small bowel is to provide surgical correction of the condition when the patient has reached the peak of general systemic improvement. Of course, some of these patients seek hospital care quite early in their illness, often permitting immediate surgery, but their number in municipal institutions is notably small. Even this group, however, receives what might be called prophylactic treatment for shock, intubation, suction and blood or plasma before and during operation, despite their apparently satisfactory condition on admission. The other, much larger, group demands an exhaustive study to assess its ability to counter the shocking effect of the

TABLE 2 (*Concluded*).

TYPE OF LESION	NO. OF CASES OPERATED ON	NO. OF CASES WITH STRANGULATION	NO. OF CASES RESECTED	OPERATIVE DEATHS	TOTAL DEATHS	MORTALITY	
						OPERATIVE	HOSPITAL
						%	%
Hernia:							
Umbilical	8	5	2	1	1	13	13
Inguinal	23	12	2	1	2	4	8
Femoral	5	5	1	1	1	20	20
Postoperative	3	1	0	0	0	0	0
Internal	5	5	1	0	0	0	0
Postoperative adhesions	22	2	1	1	1	5	2
Inflammatory adhesions	6	1	0	1	1	17	11
Intussusception	5	5	0	0	0	0	0
Volvulus	5	5	1	0	0	0	0
Mesenteric vascular occlusion	2	2	0	2	3	100	100
Carcinoma:							
Metastasis	1	0	1	0	1	0	0
Primary malignancy	1	0	1	0	0	0	0
Meckel's diverticulum	1	0	1	0	0	0	0
Regional ileitis	2	0	2	0	0	0	0
Tuberculosis of ileum	1	0	1	0	0	0	0
Totals	90	43	14	7	10		
Percentages						7.7	8.4

contemplated operation. It is difficult to make blanket recommendations for judging this ability, but generally speaking we favor postponing surgery until the pulse is stabilized below 110; the blood pressure is at normal or near-normal limits; the acid-base balance is restored, as estimated by the type of respiration, the plasma carbon dioxide combining power, acetonuria, dermination of the serum sodium and chloride levels and so forth; hydration has been achieved, as determined by the appearance and feel of the tongue, blood urea nitrogen studies, the specific gravity and quantity of urine passed during preparation and hematocrit studies; the definitive treatment of complications, such as pneumonia and heart failure, is well under way; there is significant improvement in distention; and the general effects of shock — sweating, pallor or cyanosis, coldness of the extremities and mental torpor or severe anxiety — have been alleviated.

To illustrate the importance and frequent association of these factors in the present series, certain facts are of interest. Of the 90 patients undergoing operation, only 9 (10 per cent) were admitted with pulse rates below 90. Eleven (12 per cent) were admitted with a pulse in excess of 120 and a systolic blood pressure below 100. Three (3 per cent) were admitted with no radial pulse or blood pressure detectable. Of these, 1 patient, a fifty-eight-year-old man with a strangulated inguinal hernia of eight days' duration, died. He was operated on after six hours of preparation with a systolic blood pressure of only 80 because no more blood or plasma was available. This was the only patient in this group of 11 admitted in shock who had not been restored from it before operation.

At this point there arises the question so often propounded concerning the hypothetical patient who does not respond favorably to the giving of blood or plasma, suction and so forth during the preparative period. These conditions exist very rarely when *enough* preparation is given. Even in the 3 patients with complete occlusion of the superior mesenteric artery, only 1 failed to respond favorably and was therefore not operated on. A girl of seventeen, who had had obstruction from postinflammatory adhesions for fourteen days, required 4800 cc. of blood and plasma in eighteen hours to stabilize the pulse and blood pressure and who was mistakenly declared dead by an intern shortly after admission, had an uneventful postoperative course. Even the rare patient who does not respond leaves one no alternative, however, since an anesthetic and a major operation would certainly not alleviate the state of shock.

Only 9 patients (10 per cent) were admitted within twenty-four hours of the onset of obstruction, as contrasted with 44 per cent reported by Dennis and Brown,[10] with a 7.8 per cent mortality. There was no mortality in our comparable group.

Disturbances in acid-base balance were almost the rule, being evidenced by acetonuria, depletion of serum chloride and so forth in 86 per cent of the cases tested. Major degrees of dehydration, manifested by a dry tongue, scanty and concentrated urine, excessive blood concentration and elevated blood urea nitrogen, were observed in 43 per cent of the series. Four patients (4 per cent) had pneumonia complicating the obstruction, 5 (6 per cent) had coincident cardiac failure, and 3 others had a systolic blood pressure in excess of 200.

From these data it will be seen that the recent recommendations for early or immediate operation[3, 8, 13, 14, 16, 21, 22] could not be applied to the type of patient seen in this hospital. The operation for intestinal obstruction, however simple, is made on a patient poorly constituted to tolerate it, who requires a considerable amount of care to provide reasonable assurance of his survival.

During the period from January 1, 1943, to March 1, 1945, a total of 118 cases of small-bowel obstruction were observed (Table 2). Ten of these patients died, a mortality of 8 per cent. Ninety patients were operated on, and 7 of these died, a mortality of 8 per cent. In 14 patients resection was necessary, and 3 of these died, a mortality of 21 per cent. The operations in all cases with resection, with one exception, consisted of a primary anastomosis of the aseptic type. In 2 of the fatal cases, massive gangrene of the small bowel, caused by mesenteric vascular occlusion, was found, but resection was not performed.

Summary and Conclusions

One hundred and eighteen cases of acute complete small-bowel obstruction seen in a large municipal hospital, 90 of whom were treated operatively, are presented. Data of interest on these patients are given.

The generally high mortality in this group, from several decades past to recent reports from leading clinics, is reviewed.

The necessity for evaluation of the patient as a whole rather than placing undue emphasis on local bowel disease is stressed.

A warning note is sounded against the preponderant recent advocations of immediate operation on these severely ill patients, and a plan for evaluating the proper time for operation is presented.

The general nature of the systemic condition of these patients is summarized.

Of the 118 patients treated, 10 died, a mortality of 8 per cent. Of the 90 operated on, 7 failed to survive, an operative mortality of 8 per cent.

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EPISTAXIS*

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NOSEBLEED is of frequent occurrence, often stops spontaneously and in the great majority of cases is easily controlled. It may, however, be extremely persistent and, although seldom endangering life, may cause much anxiety to both patient and physician.

In the event of persistent or recurring attacks, a search should be made for some underlying cause, such as hemophilia, leukemia, anemia or diabetes. This type of nosebleed also frequently occurs in hypertension in elderly people and in affections caused by hemolytic streptococci. It should be regarded as a surgical condition, and the usual measures should be taken for its control, through direct pressure, cauterization or cutting off the blood supply to the bleeding point, by ligation or otherwise.

A study of the anatomy of the blood supply of the nose affords a more intelligent approach to treatment. The sphenopalatine artery is a branch of the internal maxillary, which in turn is derived from the external carotid. This artery comes through the sphenopalatine foramen, and its terminal branches are distributed over the septal and lateral walls of the nasal cavity. A large branch runs along the floor of the nose and anastomoses with the great palatine artery through the incisive foramen.

A large majority of spontaneous hemorrhages occur from the so-called "Kisselbach's area," an arteriovenous plexus on the anteroinferior portion of the cartilaginous septum. These vary from simple oozing, easily controlled by pressure, to active arterial bleeding from a small vessel.

Various means of control have been advocated, among them pressure with packs saturated with hydrogen peroxide or adrenalin, the application of salt pork, fresh tonsillar tissue, snake venom and various hemostatic preparations and the submucous injection of novocain. The use of an inflated glove finger has also been suggested.

The simplest and most efficient method, when the bleeding point can be readily seen, is direct cauterization of the anesthetized mucous membrane. Care should be taken not to burn too deeply, thus perforating the opposite nostril. In some cases of mild bleeding a crystal of chromic acid fused on a metal applicator is sufficient.

The most annoying type of bleeding comes from some point that cannot readily be seen, and for which other means of control must be adopted. Bleeding may occur from a point on the posterior surface of a septal deviation not accessible to cauterization; this can be controlled by submucous resection. In recurring bleeding from Kisselbach's area, it may be unwise to repeat cauterization, and submucous elevation of the mucoperichondrium may be necessary.

Most spontaneous hemorrhages — that is, those of the nontraumatic variety — seem to come from a point below the level of the inferior turbinate; they seldom arise from the upper portion of the nasal cavity. The postnasal plug, sometimes necessary, is extremely annoying to the patient and may lead to infection of the middle ear or sinuses; the plug should ordinarily not be left in place for more than twenty-four hours. The best method of inserting it is as follows. A soft catheter introduced through the bleeding nostril is drawn out through the mouth, and a double string is attached to the

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catheter and withdrawn through the nose, pulling the plug into place. The nostril should be tightly packed and the double string tied over a sponge; a third short string attached to the plug facilitates its removal through the mouth.

Before resorting to this method, an effort should be made to locate more accurately the source of the bleeding. As already stated, most of these hemorrhages seem to come from a point below the lower turbinate. It is difficult to pack gauze into this area, and a useful procedure is to introduce several wooden applicators wound with cotton and saturated with adrenalin or cocaine; this in itself often stops the bleeding and helps to locate its source. The applicators may be left in place for some time if desired.

Reference to the blood supply of the nose shows that the largest arterial vessel is the anastomosis between the sphenopalatine and great palatine arteries through the incisive foramen, and it is at this point that persistent hemorrhage may occur. In fact, active pulsation is often seen here, at a point inaccessible to cauterization. A method that has proved successful in meeting this situation is submucous elevation of the tissue on the floor of the nose and lower meatus. Under novocain anesthesia an incision is made at the mucocutaneous juncture, allowing the insertion of a sharp submucous elevator carrying the separation back beyond the incisive foramen, thus shutting off the blood supply. If necessary, a small gauze pack may be inserted between the soft tissues and the bone. In case the hemorrhage cannot be controlled in the manner suggested, it may be necessary to tie off the external carotid artery.

SUMMARY

Methods for the control of persistent or recurring epistaxis are reviewed.

CARDIOVASCULAR DISEASE AMONG VETERANS OF WORLD WAR II*

A Survey of 19,870 Cases

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With the technical assistance of the Office of Budget and Statistics

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THERE are many gaps in the knowledge of heart disease, the leading cause of death among adults in the United States. To follow a case of cardiovascular disease from its earliest recognized manifestations throughout the lifetime of the patient may lead to a better understanding of such disease processes. Furthermore, there is a distinct need for more information concerning the natural history of circulatory disturbances that apparently commence or exhibit their first signs and symptoms in young adults. The relation of race, sex, age, place of residence and similar factors to various types of heart disease can best be appraised by analyzing and following large numbers of cases in each disease category.

At the present time, an unusual opportunity for such a study is available to the Veterans Administration in the large groups of veterans of World War II who have been discharged from the armed forces because of cardiovascular disease. This source material is of potential value for the following reasons. First, there is available a select group of patients of military age who one or two years previously were considered physically acceptable for active military service. Second, in most cases the disease was carefully observed at its inception or evaluated during its development in military service, and the patients, as beneficiaries of the Veterans Administration, will be followed through the course of the disease to its termination. Third, the medical data available for analyses are adequate, since they include induction medical examinations, medical records of hospitalization during military service and Veterans Administration medical examinations made in outpatient departments or during periods of hospitalization following separation from military service. Lastly, these veterans live in all sections of the United States.

The present study was restricted to veterans with service-connected cardiovascular disabilities incurred in or aggravated by military service on or after December 7, 1941, who were on the Veterans Administration pension rolls up to June 30, 1944. These patients, the majority of whom have not been treated in hospitals of the Veterans Administration, comprised the largest group of cardiovascular cases among World War II veterans available for analysis.

There are three other groups of veterans with circulatory abnormalities whose cases are not included in this report and are the subjects of papers now in preparation. The first group comprises vet-

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erans whose disability rating claims were disallowed and who do not receive pensions and are considered as non-service-connected. In most of these cases, the cardiovascular disability was considered to have existed prior to induction and not to have been aggravated by military service. The second group is composed of patients, whether or not service-connected, who have received hospital care from the Veterans Administration. The third group includes reserve officers receiving retirement pay for cardiovascular disabilities.

The cardiovascular diagnoses used as a basis for this study were obtained from the reports of the disability boards of the Veterans Administration. These boards obtained the diagnoses either directly from the findings of the medical officers who had treated or observed the veterans immediately preceding their discharge from the armed forces or from subsequent physical examinations conducted by the medical officers of the Veterans Administration. Most of these examinations included detailed histories, physical examinations, roentgenograms of the chest, electrocardiograms and the routine laboratory procedures, as well as many special ones.

A preliminary survey of 19,870 World War II veterans with cardiovascular disease was undertaken to appraise some of the available material for the purpose of developing practical follow-up programs. The data obtained were analyzed from the viewpoints of age, sex and race, diagnosis and geographical distribution, and the trends revealed were considered of sufficient interest to report at this time.

Age, Race and Sex Distribution

The ages ranged from seventeen to sixty-five years, with an average of 29.5 years (Table 1). Eleven thousand, three hundred and sixty-one patients (57.2 per cent) were under thirty years of age; 6254 (31.5 per cent) were between thirty and thirty-nine and 2171 (10.9 per cent) were forty years of age or older. In 84 cases the age was not recorded. An accurate racial classification was made in 18,148 patients (Table 2). Of these, 16,065 (80.8 per cent) were Whites, 1987 (10 per cent) were Negroes, and 96 (0.5 per cent) were of other races. In the 1722 remaining cases, the racial factor was not clearly stated.

There were 19,789 men and 81 women.

Diagnoses

Valvular Heart Disease

The diagnosis of valvular heart disease with no etiologic factor designated was the most frequent one, being made in 5774 cases (29.1 per cent). The ages of these patients ranged from seventeen to sixty years, with an average of 26.4 years. Five thousand, five hundred and sixty-three patients (96.3 per cent) were under forty, 4269 (73.9 per cent) were under thirty.

The distribution of the various valvular lesions in this group was interesting. Lesions limited to the mitral valve were diagnosed in 3646 cases; there was mitral insufficiency in 2273, and a mitral stenosis, with or without insufficiency, was present in 1373. Involvement of the aortic valve alone was reported in 1029 cases; the predominating lesion was diagnosed as aortic insufficiency in 859, and as aortic stenosis in 170. Combined aortic-valve and mitral-valve lesions, including various combinations of involvement, were diagnosed in 971 cases. The diagnosis of chronic endocarditis without reference to the valves involved was made in 120 cases, and tricuspid-valve and pulmonic-valve lesions were each diagnosed in 4 cases.

Table 1. *Distribution of Cases according to Diagnosis and Age.*

Diagnosis	Age (years) 15–19	20–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60–64	65–69	Not stated	Total No. of Cases	Average Age	Age Range
														yr.	yr.
Valvular heart disease	262	2,568	1,439	773	521	124	50	3	2	1	0	31	5,774	26.4	17–60
Rheumatic fever	123	1,551	719	314	181	55	21	1	2	1	0	13	2,981	25.6	18–63
Hypertension	28	560	682	641	668	198	129	18	6	1	0	14	2,945	31.5	19–61
Peripheral vascular lesions	13	468	641	593	553	171	103	13	3	0	0	5	2,563	31.4	18–57
Arteriosclerosis	5	124	153	236	410	300	355	101	36	7	1	5	1,733	38.8	19–65
Neurocirculatory asthenia	13	360	335	247	248	33	10	1	1	0	0	7	1,255	29.1	19–57
Miscellaneous diseases	19	220	178	130	67	77	58	9	3	0	1	3	865	31.1	18–65
Nonvalvular heart disease (etiology unknown)	43	504	353	282	290	133	107	32	2	2	0	6	1,754	30.7	18–63
Totals	506	6,355	4,500	3,216	3,038	1,091	833	178	55	12	2	84	19,870	29.5	
Percentages	2.55	31.98	22.65	16.19	15.29	5.49	4.19	0.89	0.28	0.06	0.01	0.42			
Veterans on pension rolls	3,983	60,362	59,790	40,219	31,220	7,422	3,921	586	135	27	4	850	208,519	28.8	
Percentages of veterans on pension rolls	1.91	28.95	28.67	19.29	14.97	3.56	1.88	0.28	0.06	0.01	0.00	0.41			

Table 2. *Distribution of Cases according to Race and Sex.*

Race	Men No. of Cases	Men Percentage	Women No. of Cases	Women Percentage	Totals No. of Cases	Totals Percentage
Whites	15,994	80.8	71	87.7	16,065	80.8
Negroes	1,982	10.0	5	6.2	1,987	10.0
Others	95	0.5	1	1.2	96	0.5
Race not reported	1,718	8.7	4	4.9	1,722	8.7
Totals	19,789		81		19,870	
Percentages		99.6		0.4		

Rheumatic Fever

Rheumatic fever was diagnosed in 2981 cases (15 per cent). The average age was 25.6 years, the lowest average encountered in any of the groups. Two thousand, three hundred and ninety-three patients (80.0 per cent) were under thirty years of age, and 2888 (96.8 per cent) were under forty.

Rheumatic fever without any recognized or diagnosed myocardial or valvular residuals occurred in 813 cases (27.4 per cent), valvular disease in 1853 cases (62.1 per cent), and rheumatic myocarditis or pericarditis in 315 (10.5 per cent).

The distribution of lesions among the 1853 cases of valvular disease was as follows: mitral lesions alone, 1423 cases; aortic and mitral lesions combined, 197 cases; aortic lesions alone, 150 cases; and valvular disease with the involved valves not identified, 83 cases. Mitral insufficiency was diagnosed as the main valvular abnormality in 1175 cases, and mitral stenosis, with or without insufficiency, in 248 cases. Among the lesions of the aortic valve alone, the main anatomic lesion was aortic insufficiency in 128 cases and aortic stenosis in 22.

Hypertension

Hypertension was considered to be the etiologic factor responsible for the main disability of 2945 patients (14.8 per cent). Essential hypertension and hypertension with an associated cardiac disease, with or without the additional factor of coronary arteriosclerosis, are included in this category. The average age of all of the patients in this classification was 31.5 years, with a range of nineteen to sixty-one years. The large number of young people affected is noteworthy. One thousand, two hundred and seventy patients (43.1 per cent) were twenty-nine years of age or younger, and 1309 (44.4 per cent) were between thirty and thirty-nine.

Peripheral Vascular Lesions

Peripheral vascular disease other than arteriosclerosis was found in 2563 cases (12.9 per cent). Six clinical entities were included in this group, with an over-all average age of 31.4 years and an age range of eighteen to fifty-seven years. Varicose veins, thrombophlebitis and chronic phlebitis accounted for 2304 (89.9 per cent) of the peripheral vascular lesions. The remaining 259 cases were distributed as follows: thromboangiitis obliterans, 127 cases; Raynaud's disease, 108 cases; and arteriovenous fistula, 24 cases.

Arteriosclerosis

Arteriosclerosis was considered to be the etiologic factor in 1733 cases (8.7 per cent). As was expected, the average age (38.8 years) was the highest in any group studied, with a range of nineteen to sixty-five years. Nine hundred and ninety-one (57.2 per cent) of the patients were considered as having coronary arteriosclerotic heart disease and revealed an average age of 39.9 years, as compared to 37.8 years in the 742 cases diagnosed as arteriosclerosis without any definite statement concerning cardiac involvement.

The most interesting feature in this group was the large number of young men affected and the high incidence of coronary-artery occlusion. Of the 991 patients with coronary arteriosclerotic heart disease, 491 (49.5 per cent) were under forty years of age, 389 were between thirty and thirty-nine, and 235 were under thirty-five. Three hundred and fifty-seven patients (36 per cent) were diagnosed as having coronary-artery occlusion or myocardial infarction. Of these, 236 were under forty years of age and 177 were in the fourth decade, with 137 under thirty-five.

Neurocirculatory Asthenia

Neurocirculatory asthenia was diagnosed in 1255 cases (6.3 per cent). The average age was 29.1 years, and the range was nineteen to fifty-seven years. The majority of cases were in young men, with 708 (56.4 per cent) under thirty years of age and 1203 (95.8 per cent) under forty. Many cases were undoubtedly lost sight of because this syndrome was considered or diagnosed as a part of other clinical entities.

Miscellaneous Diseases

Eight diseases of the heart and blood vessels were grouped under a single classification. These affected 865 patients, whose average age was 31.1 years, with a range of eighteen to sixty-five years. This category was composed as follows: arrhythmias, 397 cases; angina pectoris, 200 cases; cerebral vascular lesions, 128 cases; pericarditis, with or without effusion, 69 cases; lesions of the aorta, 31 cases; congenital heart disease, 26 cases; acute myocarditis, 13 cases; and periarteritis nodosa, 1 case.

The etiology of the cases of arrhythmia and pericarditis was either not given or did not allow of segregation into other classifications. In the 200 cases of angina pectoris there was no mention of any associated etiologic or anatomic diagnosis. It is noteworthy that the age distribution of this small subgroup closely paralleled that of the larger arteriosclerotic group. The 128 patients with cerebrovascular lesions showed an average age of 31.5 years, with a range of nineteen to fifty-one years. There were three types of these lesions in which no etiologic factors were designated—namely, cerebral hemorrhage, 72 cases; cerebral thrombosis, 40 cases; and cerebral embolism, 16 cases. The 31 cases of aortic lesions without a definite etiologic agent were diagnosed as either aortic dilatation or aortic aneurysm. The 26 cases of congenital heart disease were considered to have been aggravated by military service. The diagnoses were as follows: patent ductus arteriosus, 7 cases; coarctation of

the aorta, 6 cases; interauricular septal defect, 2 cases; and unclassified congenital heart disease, 11 cases. The etiology was determined in all 13 cases of acute myocarditis, of which 3 were considered as due to direct trauma and 10 as secondary to identified severe infectious processes.

Nonvalvular Heart Disease of Unknown Etiology

In 1754 cases (8.8 per cent), nonvalvular cardiac damage was diagnosed without reference to the etiologic factor. Chronic myocarditis, myocardial damage and cardiac degeneration were the conditions most frequently diagnosed, occurring in 1714 cases. Cardiac hypertrophy was diagnosed in 38 cases, and cardiac dilatation in 2 cases. The average age in this group was 30.7 years, with a range of eighteen to sixty-three years.

Geographical Distribution

An analysis was made of the geographical distribution — by residence — of patients with cardiovascular disabilities, for the sole purpose of evaluating on a regional basis the follow-up potentialities of each of the discussed classifications (Table 3).

Table 3. *Distribution of Cases according to Diagnosis and Residence.*

	New England		Mid-Atlantic		Southern		Mid-Western		Western		Unknown or Outside U. S. A.		Total	
	No. of cases	Per-centages	No. of cases	Per-centages	No. of cases	Per-centages	No. of cases	Per-centages	No. of cases	Per-centages	No. of cases	Per-centages	No. of cases	Per-centages
Valvular heart disease	451	7.8	1,491	25.8	1,205	20.9	1,908	33.1	704	12.2	15	0.2	5,774	29.1
Rheumatic fever	213	7.2	578	19.4	660	22.1	985	33.0	542	18.2	3	0.1	2,981	15.0
Hypertension	135	4.6	733	24.9	875	29.7	950	32.2	250	8.5	2	0.1	2,945	14.8
Peripheral vascular lesions	204	8.0	617	24.1	601	23.4	844	32.9	292	11.4	5	0.2	2,563	12.9
Arteriosclerosis	122	7.0	493	28.5	444	25.6	432	24.9	232	13.4	10	0.6	1,733	8.7
Neurocirculatory asthenia	53	4.2	286	22.8	352	28.1	464	37.0	97	7.7	3	0.2	1,255	6.3
Miscellaneous diseases	46	5.3	207	23.9	213	24.6	294	34.0	103	12.0	2	0.2	865	4.4
Nonvalvular heart disease (etiology unknown)	92	5.3	437	24.9	505	28.8	489	27.9	226	12.9	5	0.3	1,754	8.8
Totals	1,316		4,842		4,855		6,366		2,446		45		19,870	
Percentages		6.6		24.4		24.4		32.1		12.3		0.2		
Veterans on pension rolls	6,758		57,463		55,624		64,963		22,799		912		208,519	
Percentages of veterans on pension rolls		3.2		27.6		26.7		31.2		10.9		0.4		
Percentages of population (U. S. 1940 census)		6.4		23.0		27.4		32.7		10.5				

A fairly close agreement on a percentile basis was noted between the total series of cardiovascular cases and almost all its component groups, with the total population taken as that of the 1940 national census. It must be emphasized that this distribution refers only to the home residence in June, 1944, and in no way indicates the location of the origin or development of the various disease entities.

Discussion

The grouping of the various cardiovascular conditions incorporated in this report was not entirely satisfactory. The classification was based on the information listed in the diagnoses used during the consideration of the veteran's first disability rating claim and were obtained from several sources, that is, the Army, the Navy and the Veterans Administration, each of which employed a different nomenclature. This made difficult the arrangement of such data in sharply delineated groups on the bases of etiologic or anatomical findings.

A more precise classification will be possible when the diagnoses are based on a uniform system of criteria and nomenclature. In the groups in which the etiology was not stated, many of the cases will be more appropriately designated. In the large group now considered as that of valvular heart disease, many cases will be reclassified as those of rheumatic heart disease. Obviously, practically all the cases of mitral stenosis and many of the cases in which the diagnosis was limited to aortic valvular disease and combined aortic and mitral valvular disease could be placed in the rheumatic group; this was not done in this survey unless the etiologic factor was stated in the diagnosis. In the rheumatic-fever group, the cases of actual cardiac disease will be more accurately separated from those of rheumatic fever without recognized evidence of myocardial or valvular damage; likewise, the hypertensive cases, with or without myocardial involvement, will be segregated. In any follow-up program the patients with rheumatic fever or systemic hypertension without evidence of any cardiac involvement should be classified as having potential heart disease — a separation that, at the present time, the wide variations in the nomenclature render impossible.

Study of the geographical distribution by residence showed that the various cardiovascular conditions were widely distributed throughout the Nation. This may ultimately prove of some value in apprais-

ing the effects of residence in various sections of the country on each disease entity as these cases are followed during the ensuing years.

Summary

A survey of cardiovascular disease among 19,870 veterans of World War II on the Veterans Administration pension rolls as of June 30, 1944, has been reported. All the data were obtained from the reports of the diagnoses used in the evaluation of the pension claims of veterans.

The various cardiovascular conditions were classified in eight groups, based on information available from these diagnoses, which originated from sources employing several different nomenclatures. The numbers of cases in each group were as follows: valvular heart disease with no etiologic factor mentioned, 5774 cases; rheumatic fever, with or without cardiac involvement, 2981 cases; hypertension, 2945 cases; peripheral vascular conditions, 2563 cases; arteriosclerosis, with or without cardiac involvement, 1733 cases (including 991 cases specifically designated as coronary heart disease); neurocirculatory asthenia, 1255 cases; miscellaneous diseases of the heart and blood vessels, 865 cases; and cardiac lesions other than valvular conditions, without a recognized etiologic factor, 1754 cases.

SYMPOSIUM ON MEDICAL SOCIOLOGY

HEALTH-INSURANCE PLANS*

Nathaniel W. Faxon, M.D.†

DOCTORS and hospitals cannot maintain a policy of isolationism in modern society any more than can governments and countries in the present economic world. The study of the interdependent relations that result is called "medical sociology." Today we are to explore some of the problems of medical sociology involving hospitals, but we shall soon discover that there are no watertight compartments in medical sociology, and that a study of hospital problems immediately involves doctors, patients and prospective patients.

The subject of this lecture is clear in its intent, as is the main objective — namely, better medical care at costs that can be borne. There is disagreement, however, as regards means.

Let us avoid in this connection such terms as "socialized medicine," "regimentation," "totalitarian control," "rugged individualism," "medical trust" and similar catch phrases, and stick to terms that will be enlightening without developing heated controversy.

The Present Situation

As a foundation for the consideration of this subject, it will be well to review the present situation regarding physicians, hospitals, insurance and federal or state agencies dealing with health and sickness.

Physicians are licensed by state boards to practice medicine, either individually or in groups. They may or may not be on hospital staffs. They may be on a salary basis in a medical school, hospital or industry or in a federal or state department.

Hospitals are divided into governmental hospitals — federal, state, county and city — supported by taxation; voluntary — charitable or nonprofit — hospitals supported in other ways than taxation; proprietary or private hospitals intended to be profit-making; and lastly, hospitals supported by industrial companies for the benefit of their employees. All but a few of the mental, tuberculous and chronic patients are cared for in state hospitals. Ex-servicemen in these classes are cared for in federal veterans' hospitals. There are many municipal general hospitals, and most communicable-disease hospitals are tax-supported.

There are also the United States Public Health Service and the various state departments of health, with whose duties everyone is familiar. The federal government conducts the equivalent of industrial accident insurance for federal employees. There are the Children's Bureau and its Emergency Maternity and Infant Care Program, as well as the Old-Age Insurance and Care of Crippled Children programs of the Federal Security Agency. In Massachusetts there are the Industrial Accident Board, the welfare departments of state, cities and towns, the Division of the Blind and no doubt many others.

It is thus evident that there is already an extremely wide participation by government in medical affairs and that a large amount of tax funds goes into these activities. It seems axiomatic that any further extension should be built on this framework, complementing and supplementing rather than replacing existing agencies. This is important to keep in mind.

*This is the third of a series of nine lectures on medical sociology given weekly at Harvard Medical School during January, February and March, 1945; they were sponsored by the Department of Preventive Medicine and were primarily intended for third-year students.

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Most insurance companies pay a cash indemnity to the subscriber to cover hospital costs or medical fees. The Blue Cross supplies a service payment by paying hospital charges. There is a great difference between these. The cash indemnity may or may not cover hospital charges, and in the latter case the balance of cost must be borne by the patient or the hospital. Moreover, the indemnity is paid to the individual, who may or may not pay the hospital. Service payment does cover the hospital charges up to the period of hospitalization granted, which is thirty days, with a 50 per cent reduction for ninety days more. This payment, of course, covers all but the most unusual cases. It is made to the hospital.

Basic Statements

All consideration of this subject should begin with a reading of the report of the Committee on the Costs of Medical Care, published in 1932, which presents the results of careful research and considered opinions based on them. Since the issuance of this report many other studies have been made, and out of all these have come the following generally accepted facts.

There are four groups of persons in the population. The first group includes the rich or well-to-do, who have ample resources to meet the costs of medical and hospital care, with annual incomes of $5000 or over. They form 9.2 per cent of the population. The second group is the upper middle class, with annual incomes of $2000 to $5000, forming 35.2 per cent of the population. Voluntary Blue Cross plans have appealed to this group. The third group is that of the wage earners, with annual incomes from $1000 to $2000, forming 41.0 per cent of the population. The fourth group comprises the indigent, persons with annual incomes below $1000, forming 14.0 per cent of the population.

Estimates of the cost of medical care vary, but it is somewhere between $25.30 and $66.97 for the provision of adequate medical care — that is, house, office and hospital care by physician and nurse, hospital care and to some extent dental care.

It is generally accepted that if people would pay into an insurance plan all the money they now pay to doctors, hospitals and nurses, together with what they spend on medicines, including patent medicines, full medical hospital and nursing care could be provided without further expenditure. But no one is naïve enough to believe that things will work out in this way.

On certain points there is general agreement. First, medical care is costly. Sickness is unpredictable, regarding both the persons affected and its frequency and length. There is need of some method to spread this cost, either by insurance or by taxation. Second, hospitals and doctors are concentrated in cities and scarce in rural areas. There is need to even this imbalance. Hospitals should be built in isolated places and doctors subsidized to practice there. Third, more medical care would be advantageous, especially in the field of preventive medicine. The poor get less care than they need, and the rich probably pay more for such care than they should. Fourth, medical education must be carried beyond graduation from medical school. Lastly, research must be supported.

Present Insurance Plans

Until recently, sickness has been regarded as something to be handled when it occurs. This attitude has been attacked from the medical side through the development of preventive medicine, and from the social side through the development of prepayment plans or the application of the insurance principle.

Voluntary Insurance

Commercial insurance companies. Insurance companies have for a long time sold accident insurance and, in a small way, sickness insurance with cash indemnity, the latter being handled as a sideline of life insurance. Now, however, since insurance against the costs of medical and hospital care are arousing interest, many insurance companies are offering policies similar to those of the Blue Cross and Blue Shield, except for the fact that they provide for cash indemnity.

The Workmen's Compensation Act is a form of insurance against industrial accidents and sickness.

The Blue Cross. The Blue Cross was developed to provide a means of spreading the cost of hospitalized illness through prepayment on the insurance principle. It began in Dallas, Texas, in 1932 in a limited way to provide care in the University Hospital for teachers in Baylor University, and rapidly spread to other sections of the population of that city. With the interest and support of the American Hospital Association, certain principles of operation were developed and published. There are now eighty plans in thirty-eight states, with over 16,000,000 subscribers.

The Massachusetts Blue Cross, started five years ago, has 1,000,000 subscribers. The plan is based on actuarial figures showing that of every 10 persons one is hospitalized each year for a period of slightly less than ten days — (just under eight for men and just over eight for women). It is thus estimated that for every 1000 persons insured there will be about one thousand days of hospital care.

The success of the Blue Cross is due to the fact that it meets a need in a practical way. It is a nonprofit organization and is safeguarded by coming under the direction of the State Commissioner of Insurance. The governing body is composed of sixteen directors, a majority of whom must be trustees, directors or staff members of hospitals, the remainder being representatives of the Massachusetts Medical

Society and selected representatives of industries whose employees are insured.

It is preferable to insure under the group method, since thereby no physical examination is necessary because the undesirable risks are automatically cancelled. Sixty per cent of the employees of any group of 50 or more must be enrolled; below this number a higher percentage must participate. Individual subscribers can be enrolled by submitting to a physical examination, for which they pay. The cost of membership is $10.00 a year for single persons, $19.80 for a husband and wife and $24.00 for a family, regardless of the number of children.

The Blue Cross collects money from its subscribers, issues certificates of membership, makes contracts with hospitals and pays the hospital charges of its members. The hospital makes to the Blue Cross exactly the same charges, with a few exceptions, that it would render to an ordinary patient, but with a maximum daily charge of $6 for thirty days. As previously stated, if hospitalization is continued beyond this period, the Blue Cross pays 50 per cent of the hospital charges for ninety additional days.

Blue Cross plans are quite satisfactory so far as hospitalization goes, since they are inclusive, economical and widely available.

The Blue Shield

The success of hospital-insurance plans has led to a demand for similar insurance to cover professional fees for hospital illnesses. To meet this demand, organizations have been set up and sponsored by state and other medical groups. They were started later than the Blue Cross but are now operating in thirteen states, containing 50 per cent of the population of the United States. The largest unit is in Michigan.

The Blue Shield, the name applied to the corporation founded by the Massachusetts Medical Society, was formed in June, 1943. It insures single persons with annual incomes up to $2000 and families with incomes up to $2500 and pays their doctors' fees for surgical or obstetric care in hospitals. The doctors participating in this plan accept a standard fee for operations and obstetric care. About 88,000 persons are now enrolled. The cost of membership is $10.00 a year for single persons, $19.80 for a husband and wife, and $24.00 for a family.

Blue Shield plans should and will be expanded, as rapidly as can be safely done through the gathering of actuarial experience, to include medical care and a wider range of incomes by the grading of fees.

Having seen how desirable it is to use prepayment plans for the more costly and financially crippling hospital illnesses, people are now demanding similar protection against all medical care and all sickness. Here comes the rub, for there is a wide divergence of opinion concerning what this protection will cost, estimates varying all the way from $26.00 to $66.00 a year per person.

Ross–Loos Medical Group

The Ross–Loos Medical Group, of Los Angeles, a partnership of physicians, gives 27,000 persons complete medical care in homes and in the clinic and permits ninety days' hospitalization for $30.00 a year. Obstetric and dental care and the care of venereal disease are not included. This group has had a stormy career, being opposed by the American Medical Association and suffering from financial growing pains because its actuarial figures were at first faulty.

Kaiser Permanente Foundation

The Permanente Foundation, of Henry Kaiser, an industrial-insurance plan that provides medical care in the company hospital at $6.00 a year for 62,000 employees, has recently attracted attention. The foundation plans to take families of three or more for $7.50 a month, — $90.00 a year, — or $30.00 per person, which is a reasonable sum for complete medical care.

The Group Health Association

The Group Health Association, in Washington, D. C., has also been much in evidence because of the suit brought by the United States Government against the American Medical Association.

Compulsory Insurance

Comprehensive compulsory health insurance by governmental legislation was first advanced as far back as 1916. The Workmen's Compensation Act resulted, but interest in health insurance lapsed and was not resumed until 1932, when the Social Security Bill was proposed. The medical plans therein set forth were pigeonholed, but the old-age pension and unemployment features of the bill were enacted. Ever since that time the health-insurance proposals have been revived from time to time, and these have recently been given prominence by the introduction of the Wagner–Murray–Dingell Bill. The American Medical Association has opposed all such legislation. Scrutiny of this bill has revealed many defects. Many groups have studied it, and some have made helpful suggestions concerning how such legislation can be safeguarded.

The recommendations made by a group known as the Health Program Conference are the most complete and rational of those proposed. They were published as "The Principles of a Nationwide Health Program" in November, 1944, by the Committee on Research in Medical Economics of New York, a foundation-supported organization. This program is too long to describe in detail, but its ten principles give some idea of its character. These are as follows: comprehensive coverage and service; spreading of costs; distribution of facilities according to community health requirements; encouragement of group medical practice, with hospital and

professional health centers; determination of policy through the participation of those who receive and of those who furnish service; responsibility of the medical profession for strictly medical activities; freedom of choice for physicians and patients; adequate payment of physicians and hospitals by methods that encourage quality and promote economy of service; a national system; and local administration of services under national standards.

Certain points deserve emphasis. A national system has obvious advantages. One of these is collection of payment through wage deductions, which gives benefits regardless of place of residence. Local administration is necessary because federal administration cannot adjust itself to local conditions, and because decentralization is mandatory. Control of policy should be in the hands of physicians and subscribers, not in those of a government bureau head such as the Surgeon General of the United States Public Health Service. Distinction between administrative and medical responsibilities is required, for physicians must be charged with the latter only.

The costs are to be met by contributions amounting to 12 per cent of all wages up to $3000 a year, 6 per cent to be paid by employers and 6 per cent by employees. This means that a worker earning $2000 will have to pay $120. For this sum he and his dependents will receive full medical care in the home, the doctor's office and the hospital.

* * *

The proposals for health insurance comprise three views, supported by as many different groups.

The American Medical Association opposes any national legislation, insists on individualism and advocates the extension of Blue Shield and Blue Cross plans.

The American Hospital Association proposes grants-in-aid to the states for all necessary hospital and medical care for recipients of public assistance, which strikes at the heart of an important economic problem of sickness, and the promotion of Blue Cross plans. Health and health service, it is held, are extremely personal matters. The best results are obtained when patients co-operate actively in a program of prevention and treatment that includes the payment of costs. When such participation is voluntary, a certain amount of discipline of the worker, the hospital and the doctor is involved. This condition would not exist to the same extent under a taxation plan. The Blue Cross is essentially a form of public service, yet it accomplishes its aim without public compulsion. Federal employees cannot authorize deductions for the Blue Cross. It would be well for the Government to allow this. The American Hospital Association also favors grants to states for the erection of hospitals where needed when the community cannot afford to provide them, extension and expansion of Blue Shield activities and the development of group medicine in clinics and hospitals.

The proponents of compulsory plans hold that, even granted that voluntary plans are growing, they are too slow and will never include all those who should be protected, that there will be wide differences in coverage and benefits and that a voluntary system is spotty and not inclusive. These persons believe that it will be a simple matter to extend the mechanism of the present Social Security Act to include comprehensive medical care for all wage earners throughout the country, but that this can be successfully accomplished only through compulsory legislation.

The following defects of compulsory insurance have been pointed out. People dislike compulsion. They also dislike federal control. (This can be partially overcome by decentralization through the local administration of distribution of benefits.) They fear bureaucratic development and control of doctors and hospitals and dictation as to medical care. They fear political intrusion. Lastly, they are perturbed by the increasing number of pay deductions for taxes. This plan will immediately increase these by 6 per cent, and ultimately each wage earner will pay 4 per cent more, a total of 10 per cent, which will bring the total tax payment to 35 to 40 per cent.

The present situation reduces itself to the question whether people wish to have insurance against sickness develop gradually through the extension and development of voluntary plans, such as those of the Blue Cross and the Blue Shield, which they can join if they so desire, or prefer to have federal legislation that will set up a comprehensive compulsory health-insurance plan covering all wage earners, with eventual payroll deductions of 10 per cent. The chief advantage of the first method is that, although slow, it is evolutionary and voluntary; that of the second method is that it will be immediate and comprehensive. The main disadvantage of the compulsory method is that it cannot help being cumbersome, with the inherent faults of bureaucratic control and, ultimately if not immediately, the intrusion of politics.

The object of the present lecture is not to settle this question, but to present the facts of the situation for study and evaluation.

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CASE 32031

PRESENTATION OF CASE

A fifty-one-year-old man entered the hospital complaining of severe nosebleed.

Six months before admission he first noted undue fatigability, anorexia and malaise. These symptoms increased during the following months. He was forced to quit his occupation of messenger and did a number of odd jobs, including painting. One month before admission, numerous small, red, non-elevated, nonitching, painless spots appeared over the lower legs and feet. An individual spot would persist for a time and disappear. New lesions would then develop. Three weeks before admission he was exposed to rain and cold and contracted a severe cold. His nose was obstructed, and a dry hacking cough developed. Each morning he awoke with a severe frontal headache, relieved by clearing the nasal passages. The material produced on blowing the nose resembled clots of blood, but there was no actual bleeding. He had had cramping pains in the calves and thighs, relieved by exercise. The evening before admission severe hemorrhage occurred from the right nostril and lasted for six hours. There was no bleeding from the gums. During the previous six months he had lost 16 pounds of weight.

There was no history of rheumatic or scarlet fever. Twenty-five years before admission he had been treated at this hospital for syphilis over a period of three years. Eight years before admission, the blood Hinton test was positive.

Physical examination revealed the patient to be well developed and thin; he appeared fatigued and uncomfortable. The pupils were normal. There was no epistaxis. The skin was extremely dry, with scaling over the lateral aspect of the right leg. One posterior cervical node was palpable, and one in the right axilla. A Grade III systolic and a diastolic murmur were heard over the aortic area and were transmitted upward along the great vessels. A Grade II systolic murmur was heard at the apex. The lungs were clear except for inspiratory rales at the right base. The liver was palpable three finger-breadths below the right costal margin and was firm and nontender. The spleen was firm, nontender and palpable 6 cm. below the left costal margin, 1 cm. to the left of the midline. There were numerous purpuric spots, measuring 1 to 3 mm. in diameter, over both legs up to the groin. A tourniquet test was positive.

The temperature was 102°F., the pulse 90, and the respirations 30. The blood pressure was 130 systolic, 70 diastolic.

The red-cell count was 3,700,000, with 10.5 gm. of hemoglobin. The white-cell count was 4600, with 70 per cent neutrophils, 18 per cent of which were immature. The platelets were decreased. The bleeding time was 4½ minutes, and the clotting time 45 minutes. The urine was cloudy, amber and acid, with a specific gravity of 1.022. It contained no albumin, casts or cells. The nonprotein nitrogen was 30 mg. per 100 cc. and the total serum protein 4.8 gm., with 2.8 gm. of albumin and 2.0 gm. of globulin. The prothrombin time was 21 seconds (normal, 15 to 18 seconds). The serum chloride was 98 milliequiv. per liter. A blood Hinton test was negative. Four blood cultures were negative.

An x-ray film of the chest revealed clear lung fields and a normal-sized heart. The spleen was enlarged. There were no abnormal soft masses in the abdomen. There were several calcified areas in the right upper quadrant. The skull, lumbar spine, pelvis, humeri and knees were negative.

The white-cell count varied from 2400 soon after admission to 12,600 later in the course of his disease. The temperature spiked from 99 to 103°F., with chills.

Nine days after admission the patient complained of sharp pain in the lumbar region, more marked on the right than on the left, which was aggravated by motion. Slight tenderness was elicited over the right costovertebral angle, but according to the patient the pain was more medial. X-ray examination showed no fractured rib. The abdomen was not tense, but distended with gas and tympanitic. Evidence of free fluid was present. Peristalsis was high pitched. A flat film of the abdomen revealed a considerable amount of gas in the colon and less in the lower loops of the small intestine. Repeated blood and plasma transfusions resulted in an elevation of the plasma protein to 6.4 gm. Two days later more petechiae were noted over the legs. Biopsy of an axillary lymph node revealed chronic inflammation. The ascites increased. Paracentesis yielded 2500 cc. of slightly bloody fluid, with a specific gravity of 1.008. It contained 3 lymphocytes per cubic millimeter, but no bacteria or tumor cells were seen on smear. The patient was much relieved by the procedure, but the fluid again rapidly accumulated. Gaseous distention and lumbar pain persisted. Paroxysmal sinus tachycardia appeared and the patient became dyspneic. He coughed up thick pinkish-white sputum. Shifting dullness was found in the right chest. Penicillin (32,000 units)

was given intramuscularly every two hours. The nonprotein nitrogen rose to 160 mg. per 100 cc. Large hemorrhagic patches and purpuric spots appeared over the entire body. Respiratory distress became pronounced, with hemoptysis. Rales were heard in both lungs. The pulse became slow, and the respirations more difficult. The patient expired one month after admission.

Differential Diagnosis

Dr. Charles L. Short: In summary, this is a patient with a definite past history of treated syphilis, and a positive Hinton test eight years before entry. These facts immediately introduce a disturbing element, but one that I am willing to eliminate as a primary factor in the terminal illness, provided that syphilis is not the etiology of the cardiac murmurs and valvular disease. The quality of the murmurs is not described but evidently there was a loud systolic murmur as well as a diastolic murmur at the aortic area, which was transmitted to the neck. We should like to know whether there was a thrill and whether aortic stenosis might have been diagnosed, thus suggesting rheumatic heart disease.

Perhaps the x-ray films will show whether there was dilatation of the aorta.

Dr. Clayton H. Hale: The aorta is not remarkable in this film. It does not appear widened but there is slight tortuosity. The heart itself is only slightly enlarged, without characteristic configuration. This plain abdominal film shows the large spleen and the calcified nodes.

Dr. Short: Can you see the liver?

Dr. Hale: The liver edge does not show up particularly well. There is only slight if any enlargement of the liver, however.

Dr. Short: A lot of skeletal films were taken. I suppose that we can assume that they were negative.

Dr. Hale: That is correct.

Dr. Short: And the calcified area is probably due to old tuberculous lymph nodes?

Dr. Hale: I should think so.

Dr. Short: The aortic lesion was probably nonsyphilitic. This is also favored by the negative Hinton test at the time of admission and the lack of preceding symptoms suggesting syphilitic aortitis. The skeletal x-ray films were probably taken with a view to the diagnosis of multiple myeloma, although, of course, they might have been taken with any form of malignant disease, probably metastatic, in mind. The diagnosis of multiple myeloma is a possibility, but the negative x-ray films, the lack of Bence-Jones proteinuria, the low serum protein and the absence of anything suggesting bone pain until late in the disease make it unlikely.

To go on with the summary, the patient had indefinite constitutional symptoms five months before the purpuric spots appeared. He contracted what was probably an intercurrent upper respiratory infection, and this may have been a precipitating factor in the severe epistaxis that brought him to the hospital. On examination he had the murmurs I have mentioned, purpuric spots and a few somewhat enlarged lymph nodes, one of which was biopsied and found to show only chronic inflammation. He had a palpable, possibly enlarged liver and an enlarged spleen. In addition, he had a fever of 102°F. On the laboratory side he had a moderate normochromic anemia and a low white-cell count, with the neutrophils showing a shift to the left, suggesting infection. He had normal renal function as measured by the urinary findings and the nonprotein nitrogen. Studies regarding the purpura disclosed no significant increase in the prothrombin time, an increased bleeding time and a definitely increased clotting time.

The x-ray films that we have seen are not helpful. The patient had a downhill course, with chills and a spiking fever to 103°F., the development of ascites and finally either cardiac failure or bronchopneumonia, or perhaps both. He continued to bleed and he had a terminal rise in the nonprotein nitrogen without any mention of what the urinary findings were at that time.

On reading this case over rapidly my first impression, which is sometimes but not always the best one, was that he had severe liver disease, presumably cirrhosis. The bleeding, although the prothrombin time was not definitely increased, the low white-cell count, the depressed serum albumin, the ascites, the palpable liver and the splenomegaly all seem to fit in with this diagnosis. After reading more carefully, however, I found the sentence stating that he had a fever of 103°F., with chills. This made me pause, as well as the absence of jaundice in a patient with as much bleeding and who was as sick as this. I believe, and Dr. Jones may correct me on this, that a fever of this type and duration is incompatible with the diagnosis of hepatic cirrhosis in the absence of a complicating infection. I accordingly decided against that diagnosis.

The primary or idiopathic form of purpura must, of course, be considered. As for the thrombopenic variety, the bleeding time was only slightly, if at all, increased compared to the clotting time. The clot retraction was presumably normal, and the platelets were said to be decreased, rather than absent or nearly absent. In regard to the type of idiopathic purpura that is not associated with platelet depression, cases as severe as this have been described with fatal termination, but the diagnosis is questionable. Probably many of them were cases of disseminated lupus erythematosus. And this brings up the possibility of disseminated lupus, which may be associated with severe purpura; in addition, I remember one patient who also had ascites. I do not believe, however, that there is enough positive evidence to consider that diagnosis any further.

In any obscure illness periarteritis nodosa sooner or later raises its head, and it must be thought of here. Purpura has been described in this disease, but again there are no characteristic manifestations to allow me to discuss this possibility further.

One must also mention lymphoma in passing, which was evidently considered, on account of the lymph-node biopsy. I think we can also pass by lymphoma, as well as leukemia, with the comment that positive evidence is wanting.

So far I have not mentioned any disease of known infectious origin except syphilis. Those in charge of this patient evidently thought of this possibility when the blood cultures were taken and when they finally began to treat him with penicillin. I think that a diagnosis of subacute bacterial endocarditis must have occurred to all of you, with the combination of a valvular lesion, chills, fever, anemia and an enlarged spleen. I say "a valvular lesion," although that has not been proved; but I think that we must assume that it was there and that the murmurs were not merely associated with a moderate anemia, which he had on admission to the hospital. Frank purpura with platelet depression is occasionally found in this disease. The outs about this diagnosis are the negative blood cultures, which do not eliminate it, and the development of ascites, although the latter may have been due to cardiac failure or the low serum albumin, or to a combination of the two. The lumbar pain may be explained on the basis of renal infarcts. These or likelier the glomerulonephritis found quite often in cases of subacute bacterial endocarditis may have accounted for the terminal rise in nonprotein nitrogen. So I should like to propose as a diagnosis in this case subacute bacterial endocarditis arising from rheumatic disease of the aortic valve.

Dr. Benjamin Castleman: Dr. Bland, have you an opinion?

Dr. Edward F. Bland: I agree with Dr. Short.

Dr. Wyman Richardson: This was indeed a puzzling case. I should like to say that, so far as the murmurs were concerned, Dr. Kennedy of the House Staff in one of the later notes stated that a definite mitral murmur was heard, which led to the diagnosis of a rheumatic heart. I also put down a note saying that I could not hear this murmur. You can believe either one of us.

Another puzzling thing to me was the clotting time. It was definitely prolonged, and the tendency to bleed was not due to platelet lack. Was the sedimentation rate determined?

Dr. George C. Cotzias: Yes; it was increased.

Dr. Richardson: If the increased clotting time was due to severe liver disease and fibrinogen lack, the sedimentation rate might have been slow, but I do not know whether that can be used as a yardstick. We did not measure the actual fibrinogen levels in the blood, which might have been interesting.

I wrote in the record as follows:

> I cannot easily put this picture together. There is not sufficient evidence in the blood picture to establish a diagnosis of leukemia. The platelets, although reduced, are not low enough to produce purpura; the bleeding time is normal. He has a "toxic" purpura, possibly enhanced by prothrombin lack. The heart lesion, which I think is aortic regurgitation without definite mitral stenosis, can well be syphilitic. However, I think the most likely [*sic*] diagnosis is bacterial endocarditis, with superimposed liver failure, probably cirrhosis.

Dr. Chester M. Jones: Dr. Short raised the question of fever in relation to cirrhosis. Occasionally in cirrhosis one gets prolonged fever, usually individual spikes for two or three days. It could go to that height or higher in short spikes.

Dr. Short: But not a continued fever and not with chills?

Dr. Jones: No; continuous fever is unusual. In regard to the sedimentation rate, I think that Dr. Richardson is wrong. In cirrhosis of any degree the sedimentation rate is usually greatly increased. One thing against an early type of cirrhosis is the one figure for the globulin, which was relatively low. Most advanced cases of cirrhosis tend to have normal or high globulin levels.

Clinical Diagnoses

Subacute bacterial endocarditis?
Purpura (? etiology).
Cirrhosis of liver?

Dr. Short's Diagnoses

Subacute bacterial endocarditis.
Rheumatic valvular disease, aortic, with regurgitation.
Renal infarcts?
Glomerulonephritis?

Anatomical Diagnoses

Subacute bacterial endocarditis, aortic valve.
Rheumatic heart disease, with mitral and aortic involvement.
Myocardial infarcts, small, multiple.
Portal cirrhosis of liver, mild.
Anasarca.
Infarcts of kidneys and spleen.
Bronchopneumonia.

Pathological Discussion

Dr. Benjamin Castleman: The autopsy showed what at first glance seemed to be severe cardiac failure. There was anasarca, with 3 or 4 liters of fluid in the abdominal cavity and 2 liters in each chest cavity, as well as peripheral edema. The heart, however, was normal in size and was involved with subacute bacterial endocarditis. There was old rheumatic heart disease, with mild involvement of the mitral valve and mild interadherence of the aortic cusps, but apparently not enough to produce cardiac hypertrophy. Superimposed on the old rheumatic aortic valve was a subacute bacterial

endocarditis. In this picture (Fig. 1) you can see the interadherence of the two cusps at the commissure. There, also, the part of the cusp not involved with bacterial endocarditis is thickened, showing the old rheumatic infection. You can also see the thickening of the chordae tendineae of the aortic leaflet of the mitral valve. The chordae were slightly shortened, but not enough to cause appreciable stenosis. Microscopically there were small infarcts in the myocardium due to small emboli from the aortic valve. These may be a real factor in the production of the cardiac failure seen in subacute bacterial endocarditis. This man also had infarcts in both kidneys, and I suppose the pain in the back was due to one of these infarcts. The spleen weighed over 400 gm. and showed infarction. There was a severe terminal bronchopneumonia. He had a mild portal cirrhosis, which may or may not have had anything to do with the recent illness.

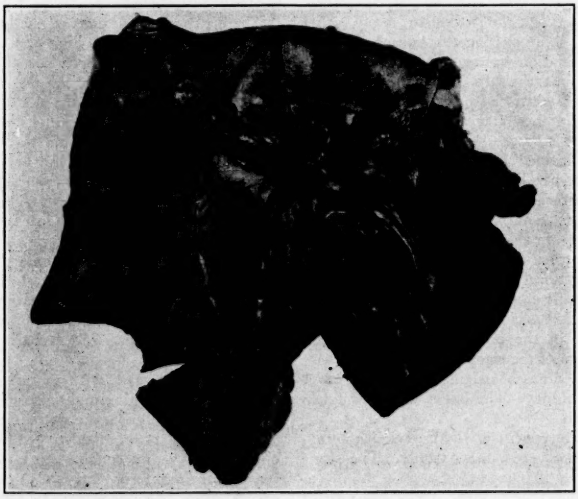

FIGURE 1. *Photograph of Heart.*

DR. RICHARDSON: Why the ascites? Was it cardiac? Since he had a low protein, I should think that one could say that the liver did have something to do with it.

DR. CASTLEMAN: It was a very mild cirrhosis, with only slight fibrosis in the portal spaces. The liver had not shrunken; in fact it was larger than normal. I had supposed that the anasarca was due to cardiac failure. It could have been due to a combination of both factors.

DR. RICHARDSON: The serum protein was certainly low.

DR. CASTLEMAN: Yes, but I do not recall having seen 2 liters of fluid in each pleural cavity as a result of a low protein.

DR. JONES: We have seen it, but the cirrhosis is usually accompanied by real liver failure. I wonder whether there was much hepatic failure.

DR. CASTLEMAN: There may have been a little, but microscopically there was only mild cirrhosis and slight chronic passive congestion.

DR. RICHARDSON: What about the increased clotting time? Can you answer that? He certainly had an increased clotting time and a tendency to bleed, and they were not due to platelet lack.

DR. CASTLEMAN: Patients with bacterial endocarditis do have a tendency to bleed. I am under the impression that some of the so-called "petechiae" seen in subacute bacterial endocarditis are not embolic but the result of capillary damage.

DR. BLAND: That is one idea, but I have never seen it happen in the presence of such an increase of the clotting time.

DR. SHORT: I have never heard of a case like this, with a bleeding tendency associated with an increased clotting time.

DR. RICHARDSON: It is usually due to a toxic purpura.

DR. BLAND: This is a disturbing case. I have looked for years for a case with this degree of heart failure and a normal-sized heart. I think that other factors should be emphasized. Were the veins distended?

DR. COTZIAS: Dr. Kennedy thought that they were not distended.

DR. BLAND: That is against heart failure.

DR. JONES: Perhaps the anasarca was due to a low serum albumin without cirrhosis. A number of cases get a low albumin due to prolonged malnutrition.

DR. SHORT: Did he have glomerulonephritis?
DR. CASTLEMAN: No.

CASE 32032

PRESENTATION OF CASE

First admission: A forty-one-year-old man, a foundry chipper by trade, entered the hospital complaining of pain about the umbilicus, radiating to the right lower quadrant and to the right testis.

Fifteen years before admission the patient had had severe right renal colic and had passed a stone. Following this attack, he had had many other episodes of renal colic, passing stones each time.

Physical examination was negative.

The red-cell count was 4,100,000, with 75 per cent hemoglobin. The white-cell count was 9100. The urine was light amber and acid, with a specific gravity of 1.020. It gave a + test for albumin, and the sediment contained 150 red cells and an occasional white cell per high-power field. The urine was sterile on culture. The nonprotein nitrogen was 23 mg. per 100 cc., and the serum protein 6.1 gm. The maximum serum calcium was 15.7 mg. per 100 cc., with a phosphorus of 2.4 mg. The alkaline phosphatase was 10.7 Bodansky units. The urinary calcium output was 489 mg. in twenty-four hours. A blood Hinton test was negative.

An x-ray film of the abdomen showed a 1-cm. stone in the region of the right kidney pelvis. There were two minute areas of calcification in the lower pole of the left kidney. The bones were normal in density.

The patient underwent a right pyelolithotomy, from which he recovered uneventfully. The stone obtained consisted of phosphatase.

Second admission (one month later). The patient was readmitted to the hospital, and an adenoma of the left lower parathyroid gland, measuring 2 by 1.5 by 1.5 cm., was removed. The patient recovered without tetany. Two days after the operation, the serum calcium was 10.3 mg. per 100 cc., the phosphorus 1.5 mg., and the alkaline phosphatase 7.1 Bodansky units. The Sulkowitch test for urinary calcium was negative at discharge.

Third admission (twenty months later). After discharge the patient felt well and passed no stones. Soon, however, he noted the gradual development of an area of hypesthesia on the anterior aspect of each thigh. Each area was small at first but then grew larger, involving the lateral aspect of the thigh as well. Five months before readmission he had a chill, and a short time later he complained of a dull, constant, nonradiating ache in the midline of the upper lumbar region, which was associated with a considerable amount of spasm of the lumbar muscles. Exertion seemed to relieve the pain somewhat, and leaning forward and to the right aggravated it. An x-ray film showed moderate scoliosis of the dorsal spine, with convexity to the right. The lumbar spine had a mild compensatory scoliosis to the opposite side. The neural arches of the first sacral segments were not united. There were hypertrophic changes throughout the spine. Two fairly large spurs arose from the lateral margins of the body of the second lumbar vertebra. In spite of a back brace and a change to a lighter type of work, the back pain persisted, occasionally waking the patient at night. It became much worse on coughing or sneezing. The patellar and Achilles tendon reflexes were diminished. There was no Babinski sign. Since the pain had become so severe, he was admitted for further study.

There was no tenderness of the lumbar vertebras at the site of the pain, which was especially severe when the patient straightened up after leaning over.

The serum calcium was 9.6 mg. per 100 cc., the phosphorus 2.4 mg. and the alkaline phosphatase 6.8 Bodansky units. The spinal fluid was negative.

An intravenous pyelogram was negative. The bones of the lumbar spine and pelvis were slightly decalcified, and there was incomplete fusion of the laminas of the sacral segments. There were moderate degenerative changes about the bodies of the first and second lumbar vertebras. A lateral film of the lumbar spine and stereo films of the sacroiliac joints revealed normal intervertebral-disk spaces. There was slight new-bone formation in the anterocentral portion of the left sacroiliac joint. A gastrointestinal series and a barium enema were negative.

The pain persisted, with variations in intensity. The hypesthesia of the thighs also continued. Several weeks after admission the patient began to expectorate about 10 cc. of blood-tinged fluid every morning. There was no cough. No bleeding point was found in the mouth or pharynx. A chest film showed normal lungs. On discharge, two months after admission, he was walking for short periods and complained infrequently of slight back pain.

Fourth admission (one year later). Three weeks after discharge the patient returned to the Out Patient Department, with marked increase in the back pain. X-ray treatment (600 r) was given over the lower lumbar vertebras and sacrum, with no relief of pain. Two days before readmission, after several hours of malaise, the patient noticed pain in the right lower quadrant of the abdomen. There had been no nausea or vomiting.

Tenderness, voluntary spasm and rebound pain were elicited in the right lower quadrant. The white-cell count was 8800.

A laparotomy was done, and an acutely inflamed appendix was removed. Convalescence was uneventful.

Fifth admission (four months later). Several weeks after operation, the pain became much worse, with radiation in a band-like manner around the anterior abdominal wall. There was considerable spasm of the sacrospinalis musculature, with limitation of flexion, extension and lateral bending of

the spine. Six weeks before readmission, the patien noticed pain in the lower anterior portion of the left chest. It was constant, sharp and localized, and was made worse by lying down or on deep breathing. Three days before admission the pain became more severe than usual, and he had shaking chills.

On physical examination the left lower portion of the chest was dull to percussion laterally and posteriorly, with absent tactile fremitus and faint breath sounds.

The temperature was 101°F., the pulse 90, and the respirations 25.

The white-cell count was 6100, with 63 per cent neutrophils. The urine was negative. The nonprotein nitrogen was 22 mg. per 100 cc., and the serum protein 7.2 gm., with 4.6 gm. of albumin and 2.6 gm. of globulin. The serum calcium was 8.6 mg. per 100 cc., the phosphorus 2.5 mg., the alkaline phosphatase 11.7 Bodansky units, and the acid phosphatase 1.2 units. The urinary calcium output was 158 mg. in twenty-four hours.

An x-ray film of the chest showed that the left lower lung field was obscured by ground-glass density. There was a linear area of increased density along the left lateral chest wall. The right lung field was clear. After thoracentesis had been done, it was found that the lung markings of the left lower lobe were crowded behind the heart. The visualized bones of the dorsolumbar spine and pelvis showed no definite evidence of metastatic involvement.

One month later only a small amount of fluid was found in the left pleural cavity, chiefly posteriorly, obliterating the left costophrenic sinus. The left lower lobe was markedly decreased in size. There was slight displacement of the heart and mediastinum to the left, and considerable emphysema of the left upper lung field. The heart and the right lung were normal. Two months after admission, narrowing of the interspace between the twelfth dorsal and first lumbar vertebras was seen. The first lumbar vertebra showed multiple areas of diminished density throughout, particularly in the anterior portion of the body. About these areas of diminished density were areas of increased density. The skull, lower legs, forearms and hands showed no bony abnormality.

Repeated thoracenteses were done, which yielded up to 1000 cc. of dark-yellow, slightly hazy fluid that contained no acid-fast bacilli or tumor cells. The patient continued to complain of lumbar pain, and also of abdominal pain radiating to the flank, with tenderness. Bronchoscopy was negative. He was discharged somewhat improved.

Sixth admission (three months later). The pain in the lower back, hips and legs had continued, being severer on the right than on the left. There were areas of numbness on the anterior and lateral aspects of each thigh. There was no pain in the neck.

Physical examination revealed dullness to percussion over the left lung base in an area about 6 cm. wide. The breath sounds were normal. There was slight tenderness over both costovertebral angles. The left knee jerk was less active than the right. The abdominal reflex was questionably absent on the right. Over the areas of subjective numbness described above, the patient was unable to distinguish between sharp and dull.

The white-cell count was 7700, with 69 per cent neutrophils. The urine was negative. The serum calcium was 11 mg. per 100 cc., the phosphorus 2.4 mg., the alkaline phosphatase 8.7 Bodansky units, and the acid phosphatase 2.4 units. A guinea pig inoculated with chest fluid obtained three months previously was killed and reported to be negative. Agglutination tests for typhoid fever and brucellosis were negative.

An x-ray film of the chest revealed no change in the appearance of the lungs. The destructive process involving the bodies of the twelfth dorsal and first lumbar vertebras had increased, with narrowing of the disk space. At the lower portion of the body of the twelfth thoracic vertebra there was a cavity apparently containing sequestrums. There was also beginning involvement of the lower surface of the first lumbar vertebra and the disk below it.

Lumbar puncture obtained clear fluid under an initial pressure equivalent to 105 mm. of water. On jugular compression the pressure rose to 350 mm. and fell slowly to 130 mm. in release; the final pressure was 90 mm. The total spinal-fluid protein was 47 mg. per 100 cc.

An operation was performed.

Differential Diagnosis

Dr. Otto E. Aufranc: It seems that, chemically, this patient improved considerably after the adenoma of the parathyroid gland was removed. So we might eliminate that entirely from the discussion.

May we see the x-ray films?

Dr. Clayton H. Hale: This chest film shows evidence of old pleurisy on the left, and the left lower lobe is collapsed. No evidence of metastases is seen, and there is no definite tuberculous lesion in the lung fields. This film of the abdomen was part of an intravenous pyelogram and shows normal kidneys. There are hypertrophic spurs of the vertebral bodies, most marked about the second lumbar vertebra. The next films, taken in the early part of this year, show definite destruction of the twelfth thoracic vertebra, and there also appears to be narrowing of the twelfth interspace. I cannot make out a definite soft-tissue mass about this vertebra, although the psoas shadow on the right in some of the films looks a little fuller than that on the left. In the differential diagnosis one must consider a malignant neoplasm, primary or metastatic, and infection. The fact that the interspace is narrowed suggests an infectious process, even though we do not have a definite soft-tissue mass to go with it.

DR. AUFRANC: We have a forty-one-year-old man who, over two years' observation, had more or less constant pain in the back. During that time x-ray studies gradually pointed out a lesion in the lumbar spine, which at the last examination consisted chiefly of destruction, with some production of bone in one film. The anterior portion of the body of the vertebra showed sequestration, so that the differential diagnosis seems to narrow down to a few things, the likeliest one of which is infection. The slow development of the destruction may well point to some malignant tumor, metastatic or primary, but the fact that he had had episodes of chills and fever seems to suggest an infectious process rather than a tumor. The radiation of pain around the thighs and to the anterior abdomen may well have been accounted for by slightly increasing abscess formation anteriorly around the bodies of the vertebras involved.

One of the most frequent types of infection at this age is tuberculosis, and the fact that this man had sequestral formation fits in with that possibility. Other types of low-grade infection, such as staphylococcal infection, typhoid fever and brucellosis, should also be considered. The gradual spread of hypesthesia and numbness is further evidence pointing to a small slowly growing abscess somewhere. I believe that the dull pain on the right side may be attributed to the operation for kidney stone. Among the other less likely infections that one might think of is echinococcal cyst, a case of which I had not seen until I was in the Army; but now that I am out, I suppose that I should forget about it.

Among the types of tumor to be considered are primary carcinoma, metastatic cancer and possibly hemangioma. If it were hemangioma, it should have shown up earlier. There was no apparent source for metastatic disease: the gastrointestinal system was negative, and the prostate and the thyroid gland, which was explored once, were negative. So more than likely we can rule out tumor.

There is one other possibility, but it may have no bearing on the case, namely, that a small sponge was left in the wound at the time of the kidney operation and that it eventually resulted in a small abscess in the body of the vertebra, indicated by destruction around the body of the vertebra. I am not sure, however, that such an abscess would produce that much bone destruction.

My diagnosis is an infection — most probably not tuberculosis because of the slow rate of onset and the absence of tubercle bacilli, although I do not know that the latter rules it out. It was possibly osteomyelitis of the spine, with sequestral formation and a small abscess. There is no note concerning what was found at the time of appendectomy or whether the area was palpated; so there might have been a mass that was missed. The second possibility is tuberculosis.

DR. MARIAN W. ROPES: This patient was a problem to us for two years. During the first part of the observation we hunted hard for a malignant tumor, since we thought that this was the likeliest explanation, but we failed to obtain any evidence other than the elevated phosphatase, which had persisted since the parathyroidectomy. The other two possibilities considered were rheumatoid spondylitis and tuberculous infection of the spine. Incidentally, the record does not describe the improvement that took place last winter, at which time he became almost symptom free and was able to work. At the time of this improvement I thought that rheumatoid spondylitis was probably the best explanation, but with the subsequent developments I thought that malignant disease or tuberculous infection were the two likeliest possibilities. The x-ray appearance never seemed quite satisfactory for either of these. The gradual development over the course of two years was surely surprising, particularly when there was no demonstrable x-ray change until the last few months.

DR. CHARLES L. SHORT: I know the diagnosis, but I might say that, on seeing the patient after one of the operations, I thought that perhaps the pleurisy that he had had might have been considered in the differential diagnosis; in that way, perhaps on the basis of probability, the correct diagnosis might have been made.

DR. RICHARDSON: To me this case represents the value of a healthy amount of ignorance. I did not know enough to be puzzled very much. I said that this patient had a lesion of the bone, that he had had it for a long time and that he had also had recurring pleurisy with effusion, all of which to me meant tuberculosis. I advised against an operative procedure because I thought that if it were tuberculosis he might develop a sinus tract and difficulty in healing; I therefore suggested therapeutic doses of radiation. I did not realize that he had already been treated by x-ray.

CLINICAL DIAGNOSES

Rheumatoid arthritis.
Metastatic malignant disease?
Tuberculous infection of spine?

DR. AUFRANC'S DIAGNOSIS

Osteomyelitis of spine?
Tuberculosis of spine?

ANATOMICAL DIAGNOSIS

Tuberculosis of spine.

PATHOLOGICAL DISCUSSION

DR. BENJAMIN CASTLEMAN: At operation the surgeon encountered caseous tuberculosis, which was obvious even on gross examination. The slides confirmed that diagnosis. The fact that we were unable to recover tubercle bacilli from the pleural effusion does not rule out tuberculosis.

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REFRESHER COURSE FOR PHYSICIANS

The attention of all physicians in Massachusetts is called to the series of review lectures that has been arranged for the Greater Boston area by the Subcommittee on Postgraduate Medical Education of the Postwar Planning Committee of the Massachusetts Medical Society in co-operation with the Massachusetts Department of Public Health. In contrast to the method employed in the outlying districts, whereby a team of instructors gives an intensive course of talks at several localities in the district three or four times a year, this series will comprise four lectures of approximately forty-five minutes each on Mondays and Wednesdays, beginning February 18 and ending May 15. The tentative program appears elsewhere in this issue of the *Journal*. That an unusual opportunity for postgraduate instruction will be provided is evidenced by those who have signified their willingness to serve as chairmen. All who take part in the program are generously donating their time.

Although this series of lectures is primarily intended for the general practitioners and returned medical officers of Greater Boston, any licensed physician may enroll, and is urged to do so; there is no registration fee. All who plan to attend should enroll either by mailing the return postcard that has already been sent to all physicians in Massachusetts or by communicating directly with the Committee on Postgraduate Medical Education, Massachusetts Medical Society, 8 Fenway, Boston 15.

CLINICAL INVESTIGATION BY THE VETERANS ADMINISTRATION

A paper published elsewhere in this issue of the *Journal* is a token of the vast amount of useful medical information in the records of the Veterans Administration that is ripe for harvesting if pains are taken in the cultivation of the field. The problems are numerous, and the numbers of disabled veterans are large in almost every category. Furthermore, there are already able and far-sighted physicians in the Veterans Administration who are anxious to utilize to the full the golden opportunity that now presents itself in the adequate follow-up study of the cases under their jurisdiction, both for the sake of the patients themselves and for the sake of new knowledge, which is certain to come if the study is well carried out.

In the cardiovascular field alone one can list five major problems that are in urgent need of concentrated follow-up study, which should be possible from the wealth of material under the care of the Veterans Administration: rheumatic fever and its effect on the heart, hypertension, early coronary heart disease, peripheral vascular diseases and neurocirculatory asthenia. Much could be learned from a detailed study of the patient under the age of forty years who is afflicted with angina pectoris or acute coronary occlusion, and this type of case

is already represented by hundreds of persons on the rolls of the Veterans Administration.

Whatever is learned from such follow-up studies of the disabled veterans will be of immediate practical use in the diagnosis and treatment of the veterans themselves: it is not at all a matter of idle academic curiosity and experimentation. Hence, anything that can be done to foster the proper development of the medical work of the Veterans Administration will improve the care of the men and women who have helped so much in saving the world from tyranny, and will simultaneously advance medical knowledge. It is hoped that such a rare opportunity will not be allowed to slip, as it did after World War I.

OBITUARY

WALTER BRADFORD CANNON

1871–1945

Many generations of Dr. Cannon's forebears — on both sides — lived in America. Both lines were characterized by initiative and courage, and men and women among them were pioneers in the westward extension of American frontiers. Dr. Cannon was thoroughly and gloriously American.

In personality he was direct, absolutely genuine without tinge of artificiality, formal only when necessary to meet the amenities of special occasions. He met persons in all walks of life from the lowliest of employees to the world's most distinguished in science and administration, confidently, simply and directly, making real and sympathetic contact by virtue of his diversified experiences, his comprehensive intelligence and his love for all human beings. Competent in highest degree to appraise character and achievement, praise often came from him, condemnation rarely — and never expressed at large. One of the world's great scientists, he was for many years one of the most beloved and revered professors at the Harvard Medical School.

Dr. Cannon was born in Prairie du Chien, Wisconsin, on October 19, 1871. Much of his boyhood was spent in Wisconsin and Minnesota in essentially rural surroundings, whence came his resourcefulness in mechanics and interpretation of natural phenomena. Simple out-of-door pursuits — canoeing, walking and mountain climbing — and working with tools remained always his chief recreational activities. The whole of Dr. Cannon's adult life was spent as a student and faculty member at Harvard. In high school at St. Paul, Minnesota, and in Harvard College he was distinguished for high scholarship and extracurricular activities. In 1906, six years after graduation from Harvard Medical School he was made George Higginson Professor of Physiology — a record in rapidity of appraisement by the medical school. Unusual too for that day was a young professor who had not been directly exposed to European influences. His early recognition by the University was understandable for he was already engaged in independently conceived research, the pursuit of which occupied him for the remaining forty years of his life and which, step by step, led him to become the world's outstanding investigator in the highest plane of physiologic research, that of the integration of many body systems in relation to the emotions.

His retirement in 1942 brought no interruption in research, only a move into the Biological Laboratories in Cambridge. For several months preceding his death, Dr. Cannon worked in Mexico City in order to collaborate with a favorite pupil, applying to research in neurophysiology of the brain information obtained from their researches on the peripheral systemic and sympathetic nervous systems.

A truly great school of physiology developed at Harvard because of Dr. Cannon's eminence as a scientist and because of those personal qualities that inspire respect and affection through inspirational guidance of pupils. More than forty persons holding professorial or equivalent rank in important universities and institutions have been definitely exposed to Dr. Cannon's personal influence in their work. As a teacher of medical undergraduates he was a powerful influence and drew many talented men into his laboratories. He became the outstanding personality in physiology in this country and was one of the greatest figures in the Harvard Medical School in the period of its greatest progress.

Years ago, after carefully weighing his opportunities for service to the medical school, he declined the deanship. Always, however, his influence and services in the councils of the medical school were great. His judgment was always detached, his decisions prompt and productive of action. He was ever ready to fight in support of his convictions.

Dr. Cannon's first bit of research was done with Professor C. B. Davenport, while an undergraduate in college. In 1898, while a second-year medical student, he began his studies of the movements of the gastrointestinal tract by means of radio-opaque substances and x-rays. He soon discovered the great effect of the emotions on the gastrointestinal movements. Continued reflective thinking on the results of each set of experiments led to his studies on the sympathetic nervous system by itself and in co-operation with the glands of internal secretion. Most of the present-day knowledge regarding the physiologic significance of the sympathetic nervous system and its integration with the activities of ductless glands is the result of research completed by him and his collaborators before his sixtieth year. Subsequent, and to him obvious, lines of research led to his great contributions on the

chemical mediation of nerve impulses, and in his last years to investigations "on the phenomena of a pace-maker in the rhythmic pulsations of the cerebral cortex."

Only World War I interrupted the continuity of his researches. The years 1917 and 1918 were spent in France and England, with a final rank of lieutenant colonel, and resulted in his brilliant studies on traumatic shock. These years were followed by an intense interest in world science and the freedom necessary for the cultivation of scientific pursuits. Since then he maintained that "a close relation exists between democracy and science" and "between liberty and democracy."

He acted on his convictions. He was the national chairman of the Medical Bureau to Aid Spanish Democracy, a member of the Joint Anti-Fascist Refugee Committee and the president of the American-Soviet Medical Society. Six months as visiting professor at Peking Union Medical College in 1935 led him later to become president of the American Bureau for Medical Aid to China and a director of the United China Relief organizations. He was foreign secretary of the National Academy of Sciences.

While a guest of the Russian Government, following his year in China, he had the courage to state his convictions regarding conditions essential for scientific progress in an address before the International Physiological Congress held in Moscow.

Another expressed conviction of Dr. Cannon's was that "it is important for science to be understood in a democracy." Hence, among his books were four published for lay readers — three based on the results of his own and his collaborator's researches and one, his last, fortunately largely biographical, a description of the life of a scientist.

The broad application of Dr. Cannon's investigations is indicated by his induction into membership in diverse special medical societies — the American Gastroenterological Association, the American Psychological Association, the American Psychopathological Association, the Association for the Study of Internal Secretions and the Société de Psychologie (Paris).

His travels, foreign lectureships, exchange professorships and attendance at medical meetings in all lands made Cannon the man as well as Cannon the scientist a truly international figure. In visits abroad, contacts that to most persons would have meant pleasant but temporary relations with Cannon resulted in permanent friendships. He had warm lifelong friends among distinguished scientists of many countries — Britain, France, Germany, Russia, Spain, Italy, the Argentine, Mexico and China. He had the broad sagacity of Benjamin Franklin and the democracy of Abraham Lincoln. His geniality, quick wit and delightful sense of humor enlivened any group he chanced to join and made him the best of companions on all occasions.

Every possible kind of scientific honor came to Dr. Cannon — from this country, the Distinguished Service Medal, from England, Companion of the Bath, and from all civilized countries, memberships in learned societies and honorary degrees.

In the fall of 1931, Dr. Cannon completed twenty-five years of service as George Higginson Professor of Physiology. The occasion was celebrated by the presentation of his portrait to the University and by afternoon and evening exercises at which the country's most distinguished physiologists and President Lowell spoke.

His achievements and honors did not change him. He remained always the approachable, honest, simple, devoted, modest gentleman, worthy of the pride of his university and his nation.

Who's Who contains a lengthy list of Dr. Cannon's degrees, honors, decorations, honorary lectureships and societies; ten of his honorary degrees, however, are not mentioned, five of them from foreign universities. Also omitted are honorary memberships in fourteen foreign scientific societies and academies.

Dr. Cannon realized more and greater satisfactions from life than come to most — in achievement, in the admiration and affection of his fellow-men and in his family. In spite of his many diversified activities related to his scientific and humanitarian activities, Dr. Cannon was a devoted family man. He also found time to give to old friends and for the cultivation of new friendships. Regretful notices of Dr. Cannon's death have come to the University from many lands.

S. B. W.

MASSACHUSETTS MEDICAL SOCIETY

DEATHS

CAVANAUGH — Mortimer T. Cavanaugh, M.D., of Great Barrington, died October 7. He was in his seventy-fourth year.

Dr. Cavanaugh received his degree from Baltimore Medical College in 1898. He was a fellow of the American Medical Association.

His widow survives.

GOODWIN — James J. Goodwin, M.D., of Clinton, died December 29. He was in his eighty-first year.

Dr. Goodwin received his degree from the Jefferson Medical College of Philadelphia in 1892. He retired in 1943. He was a member of the American College of Surgeons and the New England Surgical Society.

His widow and three sons survive.

KAPLAN — Boris Kaplan, M.D., of New Bedford, died December 25. He was in his fifty-seventh year.

Dr. Kaplan received his degree from Saratov Medical Institute, Russia, in 1918. He was a member of the National Gastroenterological Association and a fellow of the American Medical Association.

His widow and two children survive.

LEONARD — Edward D. Leonard, M.D., of Newton Centre, died December 15. He was in his sixty-first year.

Dr. Leonard received his degree from Harvard Medical School in 1913. He joined the Newton Hospital staff in 1916 and became surgeon-in-chief in 1933, holding that position until last year, when he became an emeritus member of the staff. He was a fellow of the American Medical Association and a member of the American College of Surgeons.

His widow, a son and two sisters survive.

McNAMARA — John J. McNamara, M.D., of Brockton, died January 3. He was in his seventy-fifth year.

Dr. McNamara received his degree from Tufts College Medical School in 1900. He was formerly president of the Plymouth District Medical Society. He was a member of the Massachusetts Examining Physicians Association and a fellow of the American Medical Association.

Two brothers and a sister survive.

NEW HAMPSHIRE MEDICAL SOCIETY

DEATHS

ABBOTT — Clifton S. Abbott, M.D., of Laconia, died December 10. He was in his seventy-fourth year.

Dr. Abbott received his degree from Dartmouth Medical School in 1894. He had served as president of the New Hampshire Medical Society. He was a member of the American College of Surgeons and a fellow of the American Medical Association.

A sister survives.

CUTLER — Charles H. Cutler, M.D., of Peterborough, died December 22. He was in his seventy-ninth year.

Dr. Cutler received his degree from the University of Vermont College of Medicine in 1892. He was a fellow of the American Medical Association.

His daughter and two sisters survive.

TAYLOR — Herbert L. Taylor, M.D., of Portsmouth, died October 24. He was in his sixty-ninth year.

Dr. Taylor received his degree from Jefferson Medical College of Philadelphia in 1902. He was a fellow of the American Medical Association and the American College of Surgeons.

His widow and a daughter survive.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

DISTRIBUTION OF PENICILLIN STOPPED

Effective February 1, the distribution of sodium penicillin by the Massachusetts Department of Public Health to private physicians for the treatment of gonorrhea will be temporarily discontinued, owing to the present shortage of this product.

When sufficient quantities have been accumulated to assure a surplus over the quantity needed by state institutions and co-operating clinics, distribution to private practitioners will be resumed.

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

Clinic	Date	Clinic Consultant
Worcester	January 18	John W. O'Meara
Pittsfield	January 21	Frank A. Slowick
Fall River	January 28	David S. Grice
Hyannis	January 29*	Paul L. Norton

*Day changed.

Physicians referring new patients to the clinics should get in touch with the district health officer to make appointments.

MISCELLANY

REFRESHER COURSES AT HARVARD FOR DISCHARGED MEDICAL OFFICERS

Harvard Medical School will offer a series of six-month refresher courses for medical officers discharged from the armed forces. These courses, under the supervision of Dr. Eugene C. Eppinger, assistant dean in charge of Courses for Graduates, will emphasize current scientific knowledge and theories and advances in the medical field. Instruction will be given by the regular members of the preclinical and clinical departments of the medical school. The first course is scheduled to begin on February 1.

In offering these courses, the medical school is attempting to assist physicians who, on separation from the services, wish to obtain the latest knowledge in the broad field of medicine in preparation for re-entering a civilian medical career.

CORRESPONDENCE

FACIAL EXERCISES AS AN AID IN THE TREATMENT OF ACNE

To the Editor: An important factor in the causation of acne is the accumulation of material in the sebaceous glands: the excretion does not keep pace with its formation. A blackhead forms, which sooner or later becomes infected, and a pimple results.

Acne usually occurs on the face and neck and between the shoulder blades. Sometimes it appears on the chest. It generally does not occur on the arms, abdomen, lower back, buttocks or legs. In other words the lesions are found on the parts of the body that get little exercise. The face, where it occurs most frequently, is hardly exercised at all.

It is reasonable to suppose that, if the tone of the muscles were increased and strengthened, the excretion of sebaceous material would be facilitated. Its excretion would keep pace with its formation, and there would be no chance for a blackhead to form. Also the circulation would improve, which is an additional factor in the prevention of infection.

The required exercises to improve muscular tone are simple and easy to perform. First, the lower jaw is drawn down as far as possible and the face is stretched. This is done twenty or thirty times and repeated at convenient times during the day. This also exercises, to some extent, the muscles of the neck. The second exercise is the raising and lowering of the skin of the forehead. Thirdly, the lower jaw is pushed forward and backward many times. This exercises not only the muscles of the lower jaw but also those of the neck. And, finally, the neck muscles are exercised by bending the head forward and backward and by turning it from side to side.

Obviously, this treatment is entirely harmless and can be used in conjunction with other methods. It not only appears to be an aid in the cure of acne but also betters the patient's looks, by improving the texture and color of the skin and by increasing the tone and strength of the facial muscles.

Acne is rare in this part of the country, and I have been able to experiment on only five cases. The first was that of a high-school boy of sixteen who had the worst case of acne that I have ever seen. The cheeks, forehead, chin and neck were covered with large and small pustules. The skin was greasy and rough and pasty. Treatment was begun three and a half months ago. The patient was much ashamed of the condition of his skin; he has been most co-operative and has faithfully performed the exercises. No other type of treatment was given during this period. After about three weeks the condition of his skin began to improve, and this has continued steadily since then. I saw him two weeks ago. His face was entirely free of pimples. The skin was still rough and scarred, but it had lost its pasty, greasy appearance and was firm and tight. The expression of his face had changed, and his looks were considerably improved. Acne was still present on the neck, but to a lesser degree than when I first saw him, possibly because he had not been able to exercise the muscles of his neck so efficiently as his facial muscles.

The other four cases treated by this method had moderate to mild degrees of acne. No other treatment except the exercises was employed. Although they have been using the treatment from only one to three months, all have shown definite improvement.

The success of this treatment entirely depends on the efforts of the person using it. If the exercises are not performed regularly and often for a period of months, it is of little value. It takes time to build up the tone and strength of the facial muscles, as it does with any other group of muscles in the body.

I am submitting this idea not as a claim for the cure of acne but as a useful adjunct in its treatment.

NEIL C. STEVENS
Walpole, New Hampshire

DERMATITIS FROM DDT SOLUTION

To the Editor: The public has waited impatiently for the much publicized DDT — dichloro-diphenyl-trichlorethane. Its use is, however, attended with a certain amount of risk, not so much because of the DDT itself but because of the solvents used in its preparation. The latter include crude kerosene, refined odorless kerosene, soy-bean oil, cyclohexylamine, xylene, acetone and ether. The most popular one is deodorized kerosene. This has not only the hazard of inflammability but also produces a dermatitis if it remains in contact with the skin for a sufficient length of time and under certain conditions, as shown by the following case report.

A fifty-two-year-old janitor presented a rash on the face, neck and back of hands of two weeks' duration. He showed erythema, fine vesicles and papules, the so-called "helmet type" of eruption frequently seen after exposure to irritating dust or fumes. History revealed that for several days shortly before the onset he had used as a spray a new preparation for killing insects. After each spraying episode his skin itched, until finally his face broke out in a rash. In the course of his occupation he also used floor wax. Investigation revealed that the spraying solution consisted of 5 per cent DDT, thianite and the bulk highly refined kerosene. He was given treatment and later patch tested with the following substances: the DDT solution; floor wax; pure powdered DDT; and a control. The last was prepared by applying the DDT solution to a cloth and allowing the solvent to evaporate before the cloth was applied to the skin. There was no reaction from the floor wax, pure powdered DDT or control, but there was a marked erythema and vesiculation at the site of the DDT solution, the erythema extending around the dressing for an area of over 2 cm. on either side

Although DDT apparently does not cause a dermatitis, the solvents may and in spraying these solutions persons should exercise great care and protect the exposed areas of the skin.

JOHN G. DOWNING, M.D.
520 Commonwealth Avenue
Boston 15

NEW REGULATIONS OF DEPARTMENT OF MENTAL HEALTH

To the Editor: Under authority of Section 10 of Chapter 123 of the General Laws, the Department of Mental Health hereby establishes the entire state of Massachusetts, excepting the City of Boston, as the state-hospital district for children fifteen years of age or younger.

Commitments to Walter E. Fernald State School, Wrentham State School, Belchertown State School and Monson State Hospital will remain as before.

Effective January 1, 1946, no children fifteen years of age or younger are to be admitted to any of the following state hospitals: Boston, Danvers, Foxborough, Gardner, Grafton, Medfield, Northampton, Taunton, Westborough and Worcester.

Children fifteen years of age or younger from the City of Boston may be admitted to the Boston Psychopathic Hospital for short periods of study. Facilities for prolonged care of these children will be available only at the Metropolitan State Hospital, and if the need for prolonged care is anticipated, such children should be sent directly to the Metropolitan State Hospital. Boston committing courts may commit such children directly to the Metropolitan State Hospital without a waiver.

The intent of the new plan is to permit proper segregation of children from mentally ill adults and to provide adequate educational and recreational facilities for these children.

CLIFTON T. PERKINS, M.D., *Commissioner*
Department of Mental Health
100 Nashua Street
Boston

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Peripheral Nerve Injuries: Principles of diagnosis. By Webb Haymaker, Capt., M.C., A.U.S., neuropathologist, Army Institute of Pathology, Washington, D. C., and Barnes Woodhall, Maj., M.C., A.U.S., chief, Neurosurgical Section, Walter Reed General Hospital, Washington, D. C. 8°, cloth, 227 pp., with 225 illustrations. Philadelphia: W. B. Saunders Company, 1945. $4.50.

The authors have incorporated in this work the newest information concerning their subject based on army experience. The book is intended for medical officers, as well as for medical students and civilian physicians who will be required to treat patients afflicted with nerve injuries. The volume is divided into three sections: the first discusses the principles of innervation; the second, the examination of the patient; and the third, the clinical features of plexus and peripheral nerve injuries. The text is copiously illustrated with photographs and line drawings. The book is printed on good paper, with a good type, and is easy to read. It is recommended as a reference text for all medical libraries.

Textbook of Abnormal Psychology. By Roy M. Dorcus, associate professor of psychology, University of California at Los Angeles; and G. Wilson Shaffer, dean of the College of Arts and Sciences, lecturer in psychology and professor of health and physical education, Johns Hopkins University, and psychologist, Sheppard-Enoch Pratt Hospital, Towson, Maryland. Third edition. 8°, cloth, 547 pp., with 5 plates. Baltimore: Williams and Wilkins Company, 1945. $4.00.

The publication record of this volume speaks well for its authority as a text on its subject. First printed in 1934, the second edition was published in 1939, and the third edition of 1945 is the eleventh printing of editions and reprints. The authors believe that there has not been any marked change in directional lines during the past five years and therefore have incorporated new material in the existing framework that has stood the test of time. Some of the material has been rearranged to obtain greater clarity for the student. The text has been written for advanced students in psychology, premedical students and medical students who already have had instruction in psychology. Scientific rather than popular terminology has been used wherever possible. An extensive bibliography of 833 references has been appended to the text; 365 of these have been added from the literature of the past five years. The text is well documented, and the book should serve as a reference source for all medical and university libraries.

Radiologic Examination of the Small Intestine. By Ross Golden, M.D., professor of radiology, College of Physicians and Surgeons, Columbia University, and director of the Radiological Service, Presbyterian Hospital, New York City. 4°, cloth, 239 pp., with 75 illustrations. Philadelphia: J. B. Lippincott Company, 1945. $6.00.

The material contained in this volume has been assembled over a period of many years, and the text is based on instructional courses given by the author at various radiologic society meetings. The book has been written from the viewpoint of diagnosis, and no attempt has been made to discuss the treatment of disease. A comprehensive bibliography and an adequate index have been added to the text. The volume is well printed, with a good type, on good paper, making it easy to read comfortably.

Mass Radiography of the Chest. By Herman E. Hilleboe, M.D., medical director and chief, Tuberculosis Control Division, United States Public Health Service, and professorial lecturer on tuberculosis control, George Washington University School of Medicine, Washington, D. C.; and Russell H. Morgan, M.D., medical officer-in-charge, Radiology Section, Tuberculosis Control Division, United States Public Health Service, and assistant professor of roentgenology (on leave

of absence), University of Chicago. 12°, cloth, 288 pp., with 93 illustrations. Chicago: The Year Book Publishers, Incorporated, 1945. $3.50.

The purpose of this small book is to bring together literature on mass radiography of the chest in one volume and to provide a general handbook of methods.

The History of Surgical Anesthesia. By Thomas E. Keys, M.A. With an introductory essay by Chauncey D. Leake, M.D., and a concluding chapter, "The Future of Anaesthesia," by Noel A. Gillespie, M.D. 8°, cloth, 191 pp., with 43 illustrations. New York: Schuman's, 1945. $6.00.

Major Keys has written a short history of the development of anesthesia from its earliest beginnings to the present time. Dr. Chauncey D. Leake has provided an interesting introductory essay in which will be found much material supplementing the text of Keys, and including his personal notes on anesthesia. In commenting on anesthesia collections, Dr. Leake omitted mentioning the valuable collection of the Boston Medical Library. The appendices contain a bibliography of selected references, a chronology of events relating to anesthesiology and allied subjects dating from 4004 B.C. to 1944 A.D., with a list of sources for this chronology, and a list of selected references for a history of surgical anesthesia arranged by subject and also arranged by author. In an appendix Dr. John F. Fulton discusses the Morton and Warren tracts on ether (Letheon). A comprehensive index is provided, and the text is well printed, with a good type, on good paper in the characteristic Schuman style.

The Modern Medical World: Portraits and biographical sketches of distinguished men in medicine. By Solomon R. Kagan, M.D. 8°, cloth, 223 pp., illustrated. Boston: Medico-Historical Press, 1945. $6.00.

This volume is a continuation of a series of other volumes on medical biography written by Dr. Kagan. Although there is an index of names, an alphabetical arrangement of the text would have been more useful than the present haphazard set-up. The selection of names contains many not commonly found in medical literature. This volume should be useful in all historical collections.

Yellow Magic: The story of penicillin. By J. D. Ratcliff. 12°, cloth, 173 pp. New York: Random House, 1945. $2.00.

In this popular book Mr. Ratcliff tells the story of how penicillin was discovered and developed from the laboratory stage through the stage of clinical testing and trials to the present-day commercial production. The early chapters of the book are devoted to a history of the subject. There follow special chapters on the use of penicillin in the treatment of syphilis and gonorrhea, and in war wounds. One chapter is devoted to the work of the "heartless man in Boston," Dr. Chester S. Keefer. Dr. Keefer has written a foreword, and Dr. Morris Fishbein an introduction to the text. Mr. Ratcliff is a competent authority in the field of science, and he carefully prepared himself by visiting laboratories, hospitals and manufacturing plants preparatory to writing his short manual. The manual should prove useful to all medical and public libraries as a quick reference source on the history of penicillin.

The Newborn Infant: A manual of obstetrical pediatrics. By Emerson L. Stone, M.D., associate clinical professor of obstetrics and gynecology, School of Medicine, Yale University, and attending obstetrician and gynecologist to the New Haven Hospital. Third edition, 12°, cloth, 314 pp., thoroughly revised. Philadelphia: Lea and Febiger, 1945. $3.25.

This small book, first published in 1929, was originally the outgrowth of a short selective course of lectures given to the senior class of Johns Hopkins University School of Medicine. The work has two principal objectives: to correlate and arrange in an orderly fashion a mass of data that are otherwise scattered through the medical literature and to emphasize the obstetrician's viewpoint and responsibility. The present revision aims to review concisely the accepted principles of physiology and pathology in the newborn infant and to incorporate the added knowledge of the past decade in this field of medical practice. The text is well written, has a good index and should prove useful as a resume of its subject.

The Falling Sickness: A history of epilepsy from the Greeks to the beginnings of modern neurology. By Owsei Temkin, M.D., associate professor of the history of medicine, Johns Hopkins University. 4°, cloth, 380 pp., with 7 illustrations. Baltimore: Johns Hopkins Press, 1945. $4.00.

Dr. Temkin, in this scholarly monograph, has written a history of epilepsy from the time of earliest antiquity to the time of Hughlings Jackson, about 1880. An extensive bibliography arranged alphabetically is appended to the text. The book is well printed, with a good type, on good paper and written in an interesting manner. It should be in all medical libraries and should prove interesting for public collections.

NOTICES

ANNOUNCEMENTS

Dr. Hollis L. Albright has resumed practice at 412 Beacon Street, Boston.

Dr. John J. Cincotti, recently discharged from military service, announces the opening of an office at 475 Commonwealth Avenue, Boston, for the practice of general surgery.

Dr. Robert J. Joplin, having recently returned from overseas duty with the Navy, announces the reopening of his office at 372 Marlborough Street, Boston, for the practice of orthopedic surgery.

Dr. Jost J. Michelsen announces the opening of an office at 311 Beacon Street, Boston, for the practice of neurological surgery.

Dr. Robert S. Palmer announces his return to the practice of internal medicine at 330 Dartmouth Street, Boston.

Dr. Theodore C. Pratt is resuming the practice of general surgery at 1101 Beacon Street, Brookline.

Dr. Salvatore Scelso announces the reopening of his office at 395 Commonwealth Avenue, Boston.

Dr. Grantley W. Taylor announces his release from military service and his return to private practice at 264 Beacon Street, Boston.

Dr. Winthrop Wetherbee, Jr., recently discharged from the Army, is resuming the practice of general medicine at 24 School Street, Boston.

SYMPOSIUM ON COMMUNICABLE-DISEASE CONTROL

The North Metropolitan District of the Massachusetts Department of Public Health, in co-operation with Boston University School of Medicine, will conduct a meeting "Looking Ahead in Communicable-Disease Control" on January 22 at Boston University School of Medicine, 80 East Concord Street, Boston. The morning session will be held from 10:00 a.m. to 12:00 noon, and the afternoon session from 2:00 to 4:30 p.m. The purpose of the meeting is to present to health workers and all others interested the recent advances in communicable-disease control. Discussions on biologicals, treatment with sulfa drugs and antibiotics, isolation and quarantine have been planned. In addition, a demonstration of nursing technic in the home and hospital will be presented, with a discussion by a bacteriologist and an epidemiologist.

(*Notices continued on page xix*)